

10/697,828

=> d his

(FILE 'HOME' ENTERED AT 08:53:43 ON 29 JUL 2005)

FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS, LIFESCI' ENTERED AT 08:54:06 ON 29 JUL 2005

L1 2838 S GLYCOSYL (A)TRANSFERASE?
L2 594 S (HUMAN OR MURINE) AND L1
L3 3 S "GST4ALPHA"
L4 1 DUP REM L3 (2 DUPLICATES REMOVED)
L5 7189603 S CLON? OR EXPRESS? OR RECOMBINANT
L6 213 S L2 AND L5
L7 1025751 S COLON OR INTESTINE OR (CANCER(A)TISSUE?)
L8 21 S L6 AND L7
L9 16 DUP REM L8 (5 DUPLICATES REMOVED)
L10 258785 S TRANSMEMBRANE
L11 8 S L6 AND L10
L12 7 DUP REM L11 (1 DUPLICATE REMOVED)
E ROSEN S D/AU
L13 779 S E3
E LEE J K/AU
L14 4403 S E3
E HEMMERICH S/AU
L15 129 S E3
L16 5219 S L13 OR L14 OR L15
L17 0 S L2 AND L16
L18 0 S L1 AND L16
L19 11 S GLYCOSYL (A) SULFOTRANSFERASE?
L20 7 DUP REM L19 (4 DUPLICATES REMOVED)
L21 3 S L16 AND L20

=>

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NEWS	13	APR 04	EMBASE - Database reloaded and enhanced
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NEWS	23	JUL 01	MEDICONF removed from STN
NEWS	24	JUL 07	STN Patent Forums to be held in July 2005
NEWS	25	JUL 13	SCISEARCH reloaded
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NEWS EXPRESS			JUNE 13 CURRENT WINDOWS VERSION IS V8.0, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 13 JUNE 2005
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FILE 'HOME' ENTERED AT 08:53:43 ON 29 JUL 2005

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=> file medline embase biosis biotechds scisearch hcaplus ntis lifesci
COST IN U.S. DOLLARS                               SINCE FILE      TOTAL
                                                    ENTRY      SESSION
FULL ESTIMATED COST                               0.21         0.21
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=> s glycosyl (a)transferase2
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For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).
```

```
=> s glycosyl (a)transferase?
L1      2838 GLYCOSYL (A) TRANSFERASE?
```

```
=> s (human or murine) and l1
      3 FILES SEARCHED...
L2      594 (HUMAN OR MURINE) AND L1
```

```
=> s "GST4alpha"
L3      3 "GST4ALPHA"
```

```
=> dup rem l3
```

PROCESSING COMPLETED FOR L3

L4 1 DUP REM L3 (2 DUPLICATES REMOVED)

=> d all

L4 ANSWER 1 OF 1 MEDLINE on STN DUPLICATE 1
AN 2002654554 MEDLINE
DN PubMed ID: 12218059
TI Enzymatic synthesis in vitro of the disulfated disaccharide unit of
corneal keratan sulfate.
AU Akama Tomoya O; Misra Anup K; Hindsgaul Ole; Fukuda Michiko N
CS Glycobiology Program, The Burnham Institute, La Jolla, California 92037,
USA.. takama@burnham-inst.org
NC CA71932 (NCI)
SO Journal of biological chemistry, (2002 Nov 8) 277 (45) 42505-13.
Electronic Publication: 2002-09-05.
Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200302
ED Entered STN: 20021105
Last Updated on STN: 20030207
Entered Medline: 20030206
AB Among the enzymes of the carbohydrate sulfotransferase family, human
corneal GlcNAc 6-O-sulfotransferase (hCGn6ST, also known as human
GlcNAc6ST-5/GST4beta) and human intestinal GlcNAc 6-O-sulfotransferase
(hIGn6ST or human GlcNAc6ST-3/**GST4alpha**) are highly homologous.
In the mouse, intestinal GlcNAc 6-O-sulfotransferase (mIGn6ST or mouse
GlcNAc6ST-3/GST4) is the only orthologue of hCGn6ST and hIGn6ST. In the
previous study, we found that hCGn6ST and mIGn6ST, but not hIGn6ST, have
sulfotransferase activity to produce keratan sulfate (Akama, T. O.,
Nakayama, J., Nishida, K., Hiraoka, N., Suzuki, M., McAuliffe, J.,
Hindsgaul, O., Fukuda, M., and Fukuda, M. N. (2001) J. Biol. Chemical
276, 16271-16278). In this study, we analyzed the substrate specificities of
these sulfotransferases in vitro using synthetic carbohydrate substrates.
We found that all three sulfotransferases can transfer sulfate to the
nonreducing terminal GlcNAc of short carbohydrate substrates. Both
hCGn6ST and mIGn6ST, but not hIGn6ST, transfer sulfate to longer
carbohydrate substrates that have poly-N-acetyllactosamine structures,
suggesting the involvement of hCGn6ST and mIGn6ST in production of keratan
sulfate. To clarify further the involvement of hCGn6ST in biosynthesis of
keratan sulfate, we reconstituted the biosynthetic pathway in vitro by
sequential enzymatic treatment of a synthetic carbohydrate substrate.
Using four enzymes, beta1,4-galactosyltransferase-I, beta1,3-N-
acetylglucosaminyltransferase-2, hCGn6ST, and keratan sulfate Gal
6-O-sulfotransferase, we were able to synthesize in vitro a product that
conformed to the basic structural unit of keratan sulfate. Based on these
results, we propose a biosynthetic pathway for N-linked keratan sulfate on
corneal proteoglycans.
CT Amino Acid Substitution
Animals
CHO Cells
Carbohydrate Sequence
Chimeric Proteins: ME, metabolism
*Cornea: EN, enzymology
*Disaccharides: BI, biosynthesis
Disaccharides: CH, chemistry
Hamsters
Humans
Intestinal Mucosa: EN, enzymology

*Keratan Sulfate: BI, biosynthesis
 Keratan Sulfate: CH, chemistry
 Mice
 Molecular Sequence Data
 Mutagenesis, Site-Directed
 Recombinant Proteins: ME, metabolism
 Research Support, Non-U.S. Gov't
 Research Support, U.S. Gov't, P.H.S.
 Substrate Specificity
 *Sulfates: ME, metabolism
 *Sulfotransferases: ME, metabolism
 Transfection
 RN 9056-36-4 (Keratan Sulfate)
 CN 0 (Chimeric Proteins); 0 (Disaccharides); 0 (Recombinant Proteins); 0
 (Sulfates); EC 2.8.2 (Sulfotransferases); EC 2.8.2.- (chondroitin
 6-sulfotransferase)

=> d his

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 L3 3 S "GST4ALPHA"
 L4 1 DUP REM L3 (2 DUPLICATES REMOVED)

=> s clon? or express? or recombinant

4 FILES SEARCHED...
 L5 7189603 CLON? OR EXPRESS? OR RECOMBINANT

=> s l2 and l5

L6 213 L2 AND L5

=> s colon or intestine or (cancer(a)tissue?)

6 FILES SEARCHED...
 L7 1025751 COLON OR INTESTINE OR (CANCER(A) TISSUE?)

=> s l6 and l7

L8 21 L6 AND L7

=> dup rem l8

PROCESSING COMPLETED FOR L8
 L9 16 DUP REM L8 (5 DUPLICATES REMOVED)

=> d 1-16 ibib ab

L9 ANSWER 1 OF 16 BIOTECHDS COPYRIGHT 2005 THE THOMSON CORP. on STN
 ACCESSION NUMBER: 2004-14916 BIOTECHDS
 TITLE: Novel beta 1,3-N-acetyl-D-glucosamine glycosyltransferase
 protein which transfers N-acetyl-D-glucosamine groups from
 donors to receptors, useful in detecting cancer;
 recombinant enzyme protein production via
 plasmid **expression** in host cell for use in
 disease diagnosis
 AUTHOR: NARIMATSU H; HIRUMA T; TOGAYACHI A; NISHIHARA S
 PATENT ASSIGNEE: NAT INST ADVANCED IND SCI and TECHNOLOGY; FUJIREBIO INC
 PATENT INFO: WO 2004039976 13 May 2004
 APPLICATION INFO: WO 2003-JP13957 30 Oct 2003
 PRIORITY INFO: JP 2002-315451 30 Oct 2002; JP 2002-315451 30 Oct 2002
 DOCUMENT TYPE: Patent

LANGUAGE: Japanese
OTHER SOURCE: WPI: 2004-390324 [36]
AB DERWENT ABSTRACT:

NOVELTY - A beta 1,3-N-acetyl-D-glucosamine glycosyltransferase protein (I) comprising a polypeptide having an amino acid sequence (S1) consisting of 56-402 amino acids of a fully defined 402 amino acids as given in specification, or an amino acid sequence of (S1) in which one or more amino acids are substituted, deleted or inserted, and having an activity of transferring an N-acetyl-D-glucosamine group from an donor to the receptor, is new.

DETAILED DESCRIPTION - A beta 1,3-N-acetyl-D-glucosamine glycosyltransferase protein comprising a polypeptide having an amino acid sequence (S1) consisting of 56-402 amino acids of the fully defined sequence having 402 amino acids as given in specification, or an amino acid sequence of (S1) in which one or more amino acids are substituted, deleted or inserted, and having an activity of transferring an N-acetyl-D-glucosamine group from an donor to the substrate of receptor chosen from: (i) GalNAc (ii) GlcNAc (iii) Gal (iv) Xyl (v) Fuc (vi) Man (vii) ManNAc (viii) Gal(beta)1-4 Glc; and (ix) Gal(beta)1-4GlcNAc In which (I) has neutral vicinity and requires an binary metal ion for enzyme reaction, and GalNAc is N-acetyl-D-galactosamine residue, GlcNAc is N-acetyl-D-glucosamine residue, Gal is D-galactose, Xyl is D-xylose residue, Fuc is D-fucose residue, Man is D-mannose residue, ManNAc is N-acetyl-D-mannose, - is a glycosidic linkage, and the number is the carbon number of the sugar ring in which the glycosidic linkage exists in the formula. INDEPENDENT CLAIMS are also included for: (a) a nucleic acid (II) comprising the base sequence which codes (I), or a base sequence complementary to it; (b) a vector (III) containing (II); (c) a transformed organism (IV) containing (III); (d) antibody (V) recognizing (I); and (e) hybridizing primer pair (IV) chosen from (II) which comprises a nucleic acid sequence that hybridizes under stringent conditions to the nucleic acid which consists of a base sequence complementary to the base sequence of (II).

BIOTECHNOLOGY - Preparation: (I) is obtained by cultivating (IV) in a medium such that it **expresses** (I), and recovering (I) (claimed). Preferred Protein: (I) comprises a fully defined sequence of 402 amino acids as given in specification, or an amino acid sequence which is at least 50% identical to (S1). Preferred Nucleic Acid: (II) comprises a fully defined sequence of 1209 base pairs (S2) as given in specification, a base sequence (S3) containing 166-1206 base pairs of (S2), or a base sequence complimentary to (S2) or (S3). (II) is a DNA. (II) which comprises the base sequence, which consists of 683-775 base pairs of (S2), or a sequence complimentary to it, is used as a probe and a primer, and as a cancer marker.

USE - (I) or (II) is useful for testing the presence of cancer in sample obtained from living organism, which involves determining the amount of (I) or (II) in the sample obtained from living organism, and judging the presence of cancer in sample, if the value of (I) or (II) in the sample obtained from living organism is 1.5 times more than the value of (I) or (II) assayed from the control (normal living organism), where the amount of (I) is determined using (V), and the (II) in the sample is amplified using (IV), and then assayed. The sample is derived from the large **intestine**. (II) is used as cancer marker, and as a probe or primer (claimed).

ADVANTAGE - (I) or (II) enables testing for the presence of cancer in the sample.

EXAMPLE - RNA was extracted from the **human colon cancer tissue** and healthy large **intestine** tissue. cDNA was obtained from the isolated mRNA of both the tissues, and the PCR was performed using the obtained cDNA as template, the primers having sequences 5'-gcgacgcagatgtgttcgt-3' and 5'-caattacgtcaccagcaagca-5', and the probe having the sequences 5'-tgaggaaatctccttgaggt-3'. The **expression** level of the nucleic acid encoding

1,3-N-acetyl-D-glucosamine **glycosyl transferase** in the **human colon cancer tissue** was compared with the **expression** level of the nucleic acid encoding 1,3-N-acetyl-D-glucosamine **glycosyl transferase** in the healthy large **intestine** tissue, using the quantitative real-time PCR. The results showed that the **expression** level of the nucleic acid encoding 1,3-N-acetyl-D-glucosamine **glycosyl transferase** was 7 times more in **human colon cancer tissue** when compared to the healthy large **intestine** tissue. Thus the **expression** of the nucleic acid encoding 1,3-N-acetyl-D-glucosamine **glycosyl transferase**, is useful for detecting cancer. (52 pages)

L9 ANSWER 2 OF 16 BIOTECHDS COPYRIGHT 2005 THE THOMSON CORP. on STN
DUPLICATE 1

ACCESSION NUMBER: 2002-18789 BIOTECHDS

TITLE: Use of modulator of activity of novel **glycosyl transferase** proteins, 47169/33935 proteins, for making a medicament for modulating ability of cell to affect glycosylation state of lipid or polypeptide target in cell; vector-mediated glycosyltransferase gene transfer, **expression** in host cell, antisense oligonucleotide and transgenic animal for **recombinant** protein production, drug screening and gene therapy

AUTHOR: MEYERS R; WILLIAMSON M

PATENT ASSIGNEE: MILLENNIUM PHARM INC

PATENT INFO: WO 2002040657 23 May 2002

APPLICATION INFO: WO 2000-US47575 20 Nov 2000

PRIORITY INFO: US 2000-249939 20 Nov 2000

DOCUMENT TYPE: Patent

LANGUAGE: English

OTHER SOURCE: WPI: 2002-508326 [54]

AB DERWENT ABSTRACT:

NOVELTY - Use of a modulator of the activity of an enzyme such as 47169 protein (a novel **glycosyl transferase** protein) or 33935 protein (a novel **glycosyl transferase** protein) for making a medicament for modulating the ability of a cell to affect the glycosylation state of a lipid target or polypeptide target in a cell, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following: (1) assessing (M1) if a test compound is useful for modulating at least one phenomenon (P) such as non-covalent binding between a protein and one of a cell, a virus and another protein; cell signaling, cell differentiation, tumorigenesis, cell adhesion, cell motility, cell-to-cell interaction, cell invasivity, cell proliferation, gene transcription, and an immune response, comprising: (a) adding the test compound to a first composition comprising a 603 residue 47169 polypeptide sequence (S2), or a sequence at least 90 % identical to a 492 residue 33935 polypeptide sequence (S12), both given in the specification; and (b) comparing the activity in the first composition and in a second composition that is substantially identical to the first composition, except that it lacks the test compound, whereby a difference in the activity in the first and second compositions is an indication that the test compound is useful for modulating the phenomenon; (2) making a pharmaceutical composition for modulating at least one (P), comprising: (a) selecting a test compound according to (M1); and (b) combining the test compound with a carrier in order to make the pharmaceutical composition; (3) modulating (M3) in a **human**, at least one (P) by administering pharmaceutical composition made by the method of (2) to the **human**; and (4) identifying (M4) a compound useful for modulating at least one (P), comprising: (a) contacting the test compound and a polypeptide, or a cell that **expresses** the polypeptide, where the polypeptide is encoded by a nucleic acid at least

90 % identical to a 3985, 1809, 4158, or 3044 nucleotide sequence, all given in the specification, or has at least 25 contiguous residues of (S2) or (S12); and (b) determining if the polypeptide binds with the test compound, whereby binding of the polypeptide and the test compound is an indication that the test compound is useful for modulating the phenomenon.

WIDER DISCLOSURE - (1) an isolated 47169 or 33935 polypeptide; (2) an isolated nucleic acid molecule encoding 47169 or 33935 polypeptide; (3) a host cell which contains nucleic acid molecule encoding 47169 or 33935 polypeptide; (4) an antibody which selectively binds to 47169 or 33935 polypeptide; (5) a kit comprising a compound which selectively binds to 47169 or 33935 polypeptide or selectively hybridizes to nucleic acid molecule encoding 47169 or 33935 polypeptide, and instructions for use; (6) modulating the activity of 47169 or 33935 polypeptide involves contacting the polypeptide or a cell **expressing** a polypeptide with a compound which binds to the polypeptide to modulate the activity of the polypeptide; (7) isolated nucleic acid molecules which are antisense to 47169 or 33935-encoding nucleic acid; (8) fusion proteins comprising 47169 or 33935 polypeptide fused to non-47169 or 33935 polypeptides; (9) vectors, host cells, genetically engineered host cells, nucleic acid constructs comprising (II); (10) screening for compounds that modulate the **expression** of 47169 or 33935 nucleic acid **expression** and modulating 47169 or 33935 nucleic acid **expression** using the screened compounds; (11) use of a two-dimensional array having several addresses, each of which is positionally distinguishable from each other address of the set, and each address of the set having a unique capture probe that may be a nucleic acid (a probe complementary to nucleic acid molecule encoding 47169 or 33935 polypeptide) or a polypeptide, e.g. an antibody specific for 47169 or 33935 polypeptide for analyzing the sample; (12) nucleic acid molecules that differ from nucleic acid molecule encoding 47169 or 33935 polypeptide due to degeneracy of genetic code; (13) nucleic acid molecules encoding other 47169 or 33935 family members; (14) molecular beacon oligonucleotide primer and probe molecules having a region which is complementary to 47169 or 33935 nucleic acid molecule, two complementary regions one having a fluorophore and one a quencher such that the molecular beacon is useful for quantifying the presence of nucleic acid molecule encoding 47169 or 33935 polypeptide; (15) variants of 47169 or 33935 polypeptide which functions as agonist or antagonist; (16) making a 47169 or 33935 polypeptide e.g. a peptide having non-wild-type activity or making a fragment or analog of 47169 or 33935 polypeptide involves altering the sequence of the polypeptide e.g. the substitution or deletion of one or more amino acid residues of a 47169 or 33935 polypeptide and testing the altered polypeptide for desired activity; (17) non-**human** transgenic animals in which an endogenous 47169 or 33935 gene has been altered by homologous recombination between an endogenous gene and an exogenous DNA molecule introduced into a cell of the animal; (18) population of cells from the transgenic animal; (19) novel agents identified by screening methods involving 47169 or 33935 polypeptide; (20) a set of oligonucleotides, each of which is at least partially complementary to 47169 or 33935 nucleic acid, useful for identifying single nucleotide polymorphisms; (21) a computer-readable medium provided with 47169 or 33935 sequences; (22) making a computer readable record of a sequence of a 47169 or 33935 which includes recording the sequence on a computer readable matrix; (23) assays for determining the presence or absence of a genetic alteration in 47169 or 33935 polypeptide or nucleic acid molecule; and (24) assays for determining activity of presence or absence of 47169 or 33935 polypeptides or nucleic acids.

BIOTECHNOLOGY - Preferred Method: The modulator is inhibitor of 47169 gene **expression**, where the inhibitor is a 15-nucleotide residue long antisense oligonucleotide that hybridizes under stringent conditions with a transcript (preferably mRNA) of the 47169 gene, an

antisense oligonucleotide that hybridizes under stringent conditions with a 47169 polynucleotide. It is a molecule which does not significantly affect 47169 gene **expression** in the cell, but inhibits an activity of 47169 protein, where the agent is an antibody which specifically binds with 47169 protein, or an agent that enhances **expression** of 47169 in the cell e.g., an **expression** vector encoding 47169 protein. It can be an inhibitor of 33935 gene **expression**, where the inhibitor is an antisense oligonucleotide hybridizing under stringent conditions with transcript (preferably mRNA) of the 33935 gene, an antisense oligonucleotide that hybridizes under stringent conditions with 33935 polynucleotide. The modulator is a molecule which does not significantly affect gene 33935 gene **expression** in the cell, but inhibits activity of 33935 protein, e.g. antibody binding with the 33935 protein, or an agent which enhances **expression** of 33935 in the cell. In (M1), the test compound is added to the first composition comprising polypeptides which exhibit 47169 or 33935 activity e.g. a **glycosyl transferase** activity that is the ability to transfer an N-acetylgalactosamine moiety from uridine diphosphate to a hydroxyl moiety of a serine or threonine residue of a protein. The composition preferably comprises a cell which comprises a nucleic acid encoding either a 47169 or a 33935 protein. In (M4), the polypeptide exhibits an epitope in common with a polypeptide having the amino acid sequence of (S2) or (S12).

ACTIVITY - Antidiabetic; Antiarthritic; Antirheumatic; Immunosuppressive; Antithyroid; Antiinflammatory; Dermatological; Antiinfertility; Cytostatic. No biological data is given.

MECHANISM OF ACTION - None given.

USE - For making a medicament for modulating the ability of a cell (e.g. **human** endothelial cell such as lung cell, breast cell or **colon** cell, preferably a tumor cell) to affect the glycosylation state of a target such as lipid or polypeptide. (M3) is useful for modulating (P) in a **human** (claimed). (M3) is useful for treating disorders such as diabetes mellitus, hypoglycemia, arthritis, rheumatism, autoimmune disorders (e.g. systematic lupus erythematosus, Grave's disease, myasthenia gravis, insulin resistance, rheumatoid arthritis, scleroderma and autoimmune infertility), tumorigenesis and tumor metastasis.

ADMINISTRATION - Modulator compounds are administered by parenteral, e.g. intravenous, intradermal, subcutaneous, oral (e.g., inhalation) route etc. Dosage of modulators range from 1 micro-g-500 mg/kg, preferably 1-50 micro-g/kg.

EXAMPLE - No relevant example is given. (153 pages)

L9 ANSWER 3 OF 16 BIOTECHDS COPYRIGHT 2005 THE THOMSON CORP. on STN

ACCESSION NUMBER: 2002-18788 BIOTECHDS

TITLE: Isolated 47476, 67210, 49875, 46842, 33201, 83378, 84233, 64708, 85041 or 84234 polypeptides, useful as reagents or targets for treating or diagnosing pain or metabolic, liver, kidney, or cardiovascular disorders;

recombinant protein production, antisense, antibody and drug screening useful for gene therapy, diagnosis, mapping and pharmacogenetics

AUTHOR: MEYERS R E; CURTIS R A J; GLUCKSMANN M A

PATENT ASSIGNEE: MILLENNIUM PHARM INC

PATENT INFO: WO 2002040656 23 May 2002

APPLICATION INFO: WO 2000-US45291 14 Nov 2000

PRIORITY INFO: US 2000-250327 30 Nov 2000

DOCUMENT TYPE: Patent

LANGUAGE: English

OTHER SOURCE: WPI: 2002-508325 [54]

AB DERWENT ABSTRACT:

NOVELTY - An isolated 47476, 67210, 49875, 46842, 33201, 83378, 84233, 64708, 85041 or 84234 polypeptide (I) which comprises a fully defined

sequence of 673, 349, 600, 834, 351, 485, 320, 461, 765, 376 (S1-S10) amino acids, respectively as given in the specification, is new.

DETAILED DESCRIPTION - An isolated 47476, 67210, 49875, 46842, 33201, 83378, 84233, 64708, 85041 or 84234 polypeptide (I) which comprises a fully defined sequence of 673, 349, 600, 834, 351, 485, 320, 461, 765, 376 (S1-S10) amino acids, respectively as given in the specification, where: 47476 polypeptide is a member of ras guanine nucleotide dissociation stimulator family of proteins; 67210 polypeptide is a member of **glycosyl transferase** family; 49875 polypeptide is a member of DEAD type helicase family; 46842 polypeptide is a member of centaurin family; 33201 polypeptide is a member of dehydrogenase/reductase family, and 83378, 84233, 64708, 85041 or 84234 polypeptides are members of metal transporter family. INDEPENDENT CLAIMS are also included for the following: (1) an isolated nucleic acid molecule (II) which encodes (I), and comprises a nucleotide sequence having a fully defined sequence of 3134, 2022, 1778, 1050, 2704, 1803, 2737, 2505, 1718, 1056, 1827, 1458, 2165, 963, 2130, 1386, 3304, 2298, 2637 or 1131 nucleotides as given in the specification; (2) a host cell (III) which contains (II); (3) an antibody or its antigen-binding fragment (IV) which selectively binds to (I); (4) preparation of (I); (5) detecting (M1) presence of (II) in a sample involves contacting the sample with a nucleic acid probe or primer which selectively hybridizes to the nucleic acid molecule and determining whether the nucleic acid probe or primer binds to the nucleic acid molecule in the sample; (6) a kit comprising a compound which selectively binds to (I) or selectively hybridizes to (II), and instructions for use; (7) modulating the activity of (I) involves contacting the polypeptide or a cell **expressing** a polypeptide with a compound which binds to the polypeptide to modulate the activity of the polypeptide; and (8) inhibiting (M2) aberrant activity of 47476, 67210, 49875, 46842, 33201, 83378, 84233, 64708, 85041 or 84234-**expressing** cell with a compound that modulates activity or **expression** of (I); and (9) treating or preventing (M3) disorder characterized by aberrant activity of 47476, 67210, 49875, 46842, 33201, 83378, 84233, 64708, 85041 or 84234-**expressing** cell in a subject by administering a compound that modulates activity or **expression** of (II).

WIDER DISCLOSURE - The following are disclosed: (1) nucleic acid molecules that are substantially identical to (II), and amino acid sequences that are substantially identical to (I); and isolated nucleic acid molecules which are antisense to (II); (2) fusion proteins comprising (I) polypeptide fused to non-(I) polypeptides; (3) nucleic acid constructs comprising (II); (4) screening for compounds that modulate the **expression** of (II), and modulating nucleic acid **expression** using the screened compounds; (5) a two dimensional array having several addresses, each of which is positionally distinguishable from each other address of the set, and each address of the set having a unique capture probe that may be a nucleic acid (a probe complementary to (II)) or a polypeptide, e.g., an antibody specific for (I); (6) use of the array for analyzing the sample; (7) nucleic acid molecules which are at least 65% homologous to (II); (8) fragments of (II); (9) nucleic acid molecules that differ from (II) due to degeneracy of the genetic code; (10) molecular beacon oligonucleotide primer and probe molecules with a region which is complementary to (II), two complementary regions one having a fluorophore and one a quencher such that the molecular beacon is useful for quantitating the presence of (II); (11) variants of (I) which function as agonist or antagonist; (12) making (I) e.g., a peptide having non-wild-type activity or making a fragment or analog of (I) involves altering the sequence of the polypeptide e.g., the substitution or deletion of one or more amino acid residues of (I) and testing the altered polypeptide for desired activity; (13) a nucleic acid (N) which encodes (IV); (14) vectors including (N) and cells transformed with (N) which are useful for producing the antibody; (15) hybridomas which make (IV) and methods of using the cells

for making (IV); (16) non-human transgenic animals in which an endogenous (II) has been altered by homologous recombination between an endogenous gene and an exogenous DNA molecule introduced into a cell of the animal; (17) population of cells from the transgenic animal; (18) novel agents identified by screening methods involving (I); (19) a computer medium having executable code for receiving a subject **expression** profile; accessing a database of reference **expression** profile and either selecting a matching reference profile most similar to the subject **expression** profile or determining at least one comparison score for the similarity of the subject **expression** profile to at least one reference profile; (20) a computer medium having several digitally encoded data records each of which includes a value representing the level of **expression** of (I) in a sample and a descriptor of the sample; (21) evaluating a test compound by contacting a test compound to the cell, obtaining a subject **expression** profile (which indicates a value representing (I) **expression** level) for the contacted cell and comparing the subject **expression** profile to one or more reference profiles; (22) evaluating a subject involves obtaining sample from a subject, determining subject **expression** profile for the sample, comparing the subject **expression** profile to one or more reference **expression** profiles and selecting the reference **expression** profile most similar to the subject reference profile, where both the subject and the reference profiles include a value representing a level of (I) **expression**; (23) an array having several addresses, each of which includes a unique polypeptide and at least one address of the array has a (I) disposed on it; (24) a set of oligonucleotides, each of which is at least partially complementary to (I), useful for identifying single nucleotide polymorphisms; (25) a machine-readable medium e.g., a magnetic, optical, chemical or mechanical information storage device provided with (I) or (II) sequences; (26) making a computer readable record of a sequence of (I) or (II) which includes recording the sequence on a computer readable matrix; (27) a machine-readable medium for holding instructions for determining whether a subject has a (I)-associated disease or disorder or a predisposition to (I)-associated disease or disorder; (28) an electronic system and/or a network for determining whether a subject has a (I)-associated disease or disorder or a predisposition to (I)-associated disease or disorder; (29) a network for determining whether a subject has a (I)-associated disease or disorder or a predisposition to (I)-associated disease or disorder; (30) determining whether a subject has a (I)-associated disease or disorder or a predisposition to (I)-associated disease or disorder using the above network; and (31) assays for determining the presence or absence of a genetic alteration in (I) or (II) polypeptide or nucleic acid molecule.

BIOTECHNOLOGY - Preparation: (I) is prepared by standard **recombinant** techniques (claimed). Preferred Nucleic Acid: (II) further comprises vector nucleic acid sequences, and also comprises a nucleic acid sequence encoding a heterologous polypeptide. Preferred Polypeptide: (I) further comprises heterologous amino acid sequences. Preferred Method: In (M1), the presence of (II) is detected in a sample comprising mRNA molecules, by contacting the sample with a nucleic acid probe. In (M2), a peptide, phosphopeptide, small organic molecule or an antibody is contacted with (I)-**expressing** cells, such that the aberrant activity of a cell (preferably pre-cancerous or cancerous tissue) is reduced or inhibited.

ACTIVITY - Immunosuppressive; Cytostatic; Neuroprotective; Virucide; Antiinflammatory; Analgesic; Cardiant. No biological data is given.

MECHANISM OF ACTION - Activity or **expression** of 47476, 67210, 49875, 46842, 33201, 83378, 84233, 64708, 85041 or 84234 protein or nucleic acid, modulator; Gene Therapy.

USE - (I) is useful for identifying a compound which binds to it which involves contacting a polypeptide or a cell **expressing** a

polypeptide with a test compound and determining whether the polypeptide binds to the test compound. (III) is useful for preparing (I) by **recombinant** techniques. (IV) is useful for detecting the presence of (I) in a sample which involves contacting the sample with a compound (i.e., (IV)) which selectively binds to (I) and determining whether (IV) binds to the polypeptide in the sample. (M3) is useful for treating a disorder characterized by aberrant activity of 47476, 67210, 49875, 46842, 33201, 83378, 84233, 64708, 85041 or 84234-**expressing** cell in a subject (all claimed). 47476 molecules can act as novel diagnostic targets and therapeutic agents for controlling aberrant or deficient signal transduction resulting, in e.g., hematopoietic disorders, including e.g., blood clotting disorders, autoimmune disorders, or disorders related to an inability to clear infections (e.g., viral or bacterial infections), as well as disorders related to abnormal cellular proliferation or differentiation, e.g., leukemia. 67210 molecules can act as novel diagnostic targets and therapeutic agents for controlling disorders of metabolic imbalance (e.g., disorders of lipopolysaccharide biosynthesis or glycogen synthesis) immunological disorders (e.g., autoimmune disorders or disorders associated with an inability to clear an infection e.g., a viral or bacterial infection), cardiovascular disorders, neurological disorders, or cellular proliferation and/or differentiation disorders, e.g., cancer. 49875 molecules can act as novel diagnostic targets and therapeutic agents for controlling disorders of proliferation and/or differentiation e.g., cancer or immunological disorders. 46842 molecules can act as novel diagnostic targets and therapeutic agents for controlling cell motility and adhesion disorders (e.g., a metastatic disorder or an immunological disorder e.g., disorder related to an inability to clear an infection, e.g., a bacterial or viral infection), and secretory disorders (e.g., a neurological or viral disorder). 33201 molecules can act as novel diagnostic targets and therapeutic agents for controlling one or more disorders, including metabolic disorders, liver disorders, kidney disorders, digestive disorders, and cellular proliferative and/or differentiative disorders. 47476, 67210, 49875, 46842, 33201, 83378, 84233, 64708, 85041 or 84234 molecules can act as novel diagnostic targets and therapeutic agents for controlling one or more of cellular proliferative and/or differentiative disorders, immunological disorders (e.g., inflammatory disorders), red blood cell disorders, viral diseases, neurological disorders (e.g., brain disorders), pain or metabolic disorders, liver disorders, kidney disorders, disorders of the small **intestine**, disorder of metal ion imbalance, protein trafficking disorders, cardiovascular disorders, and disorders associated with bone metabolism. (II) is useful for **expressing** (I) via a **recombinant expression** vector in a host cell, in gene therapy applications, to detect (I) mRNA, or genetic alteration in (II) and to modulate (I) activity. Fragments of (II) are useful as probes and primers. Portions or fragments of (II) are useful to: (i) map their respective genes on a chromosome, e.g., to locate gene regions associated with genetic disease or to associate 47476, 67210, 49875, 46842, 33201, 83378, 84233, 64708, 85041 or 84234 with a disease; (ii) identify an individual from a minute biological sample (tissue typing); and (iii) aid in forensic identification of a biological sample. (II) can be inserted into vectors and used as gene therapy vectors. (I), (II) and (IV) are useful for screening assays, predictive medicine (e.g., diagnostic assays, prognostic assays, monitoring clinical trials, and pharmacogenetics); and methods of treatment (e.g., therapeutic and prophylactic).

ADMINISTRATION - Pharmaceutical compositions comprising (I), (II), (IV) are administered by parenteral, e.g., intravenous, intradermal, subcutaneous, oral, e.g., inhalation, transdermal, transmucosal or rectal routes. Dosage of (I) ranges from 0.001-30 (preferably, 5-6) mg/kg body weight. (IV) is administered in dosages ranging from 0.1-10 mg/kg body weight. Modulator compounds identified using (I) are administered in

dosages ranging from 1 microg-500 mg/kg (preferably, 1-50 microg/kg body weight).

EXAMPLE - No relevant example is given. (298 pages)

L9 ANSWER 4 OF 16 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
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ACCESSION NUMBER: 2002308977 EMBASE
TITLE: Blood group A glycosyltransferase occurring as alleles with high sequence difference is transiently induced during a *Nippostrongylus brasiliensis* parasite infection.
AUTHOR: Olson F.J.; Johansson M.E.V.; Klinga-Levan K.; Bouhours D.; Enerback L.; Hansson G.C.; Karlsson N.G.
CORPORATE SOURCE: G.C. Hansson, Dept. of Medical Biochemistry, Goteborg University, P.O. Box 440, SE-405 30 Gothenburg, Sweden. gunnar.hansson@medkem.gu.se
SOURCE: Journal of Biological Chemistry, (26 Apr 2002) Vol. 277, No. 17, pp. 15044-15052.
Refs: 35
ISSN: 0021-9258 CODEN: JBCHA3
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20020926
Last Updated on STN: 20020926

AB Neutral mucin oligosaccharides from the small **intestine** of control rats and rats infected with the parasite *Nippostrongylus brasiliensis* were released and analyzed by gas chromatography-mass spectrometry. Infected animals **expressed** seven blood group A-like structures that were all absent in the control animals. The blood group A nature of these epitopes was confirmed by blood group A reactivity of the prepared mucins, of which Muc2 was one. Transferase assays and Northern blotting on small **intestines** from infected animals showed that an α -N-acetylgalactosaminyltransferase similar to the **human** blood group A **glycosyl-transferase** had been induced. The **expression** was a transient event, with a maximum at day 6 of the 13-day- long infection. The rat blood group A glycosyltransferase was **cloned**, revealing two forms with an amino acid similarity of 95%. Both types had blood group A transferase activity and were probably allelic because none of 12 analyzed inbred strains carried both types. The second type was found in outbred rats and in one inbred strain. First generation offspring of inbred rats of each type were heterozygous, further supporting the allelic hypothesis. The transient induction and the large allelic variation could suggest that glycosyltransferases are part of a dynamic system altering mucins and other glycoconjugates as a protecting mechanism against microbial challenges.

L9 ANSWER 5 OF 16 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN

ACCESSION NUMBER: 2002:176507 BIOSIS
DOCUMENT NUMBER: PREV200200176507
TITLE: Shiga toxin binding correlates with P1 blood group antigen in **human** endothelial cells and erythrocytes.
AUTHOR(S): Gillard, B. K. [Reprint author]; Soderland, C.; McCluer, R. H. [Reprint author]; Cleary, T. G. [Reprint author]
CORPORATE SOURCE: Univ Texas Houston Medical School, Houston, TX, USA
SOURCE: Abstracts of the General Meeting of the American Society for Microbiology, (2001) Vol. 101, pp. 93. print.
Meeting Info.: 101st General Meeting of the American Society for Microbiology. Orlando, FL, USA. May 20-24, 2001. American Society for Microbiology.
ISSN: 1060-2011.

DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 6 Mar 2002
Last Updated on STN: 6 Mar 2002

AB Intestinal infection with E. coli that produce Shiga toxin (Stx) is a major health problem in the United States. Five to eight percent of infected children develop hemolytic uremic syndrome (HUS), the leading cause of renal failure in children. There is currently no way to predict which children will progress to HUS. The primary targets for Stx injury are the microvascular endothelial cells of the **colon**, kidney glomerulus and CNS. In vitro, endothelial cells from individual donors vary widely in their sensitivity to Stx. Stx binds cell surface glycolipids with terminal Gal α 1-4Gal structure and, after transport to the endoplasmic reticulum, inhibits protein synthesis. The glycolipids that bind Stx, Gb3Cer, Gal2Cer, P1 and Gal2Gb4Cer, include members of the P and LKE blood group systems. In **humans**, genetic polymorphisms in the allelic genes for the various **glycosyl-transferase** enzymes that synthesize the P and LKE blood group glycolipids have been shown to determine the amounts of these glycolipids in erythrocytes. These blood group antigens are also **expressed** in endothelial cells. As a first step in defining the role of blood group phenotype in sensitivity to Stx injury, we analyzed Stx binding to **human** erythrocytes and microvascular dermal foreskin endothelial cells by flow cytometry. As reported previously, P1 adult donor erythrocytes bound significantly more Stx than did P2 erythrocytes. Similarly, P1 newborn cord erythrocytes, obtained from donors of microvascular endothelial cells, showed 5-fold higher median fluorescence intensity for Stx binding than did P2 cord erythrocytes (p=0.003). Most importantly, in microvascular endothelial cells Stx binding significantly correlated with P1 **expression** (p=0.024). Our results demonstrate that P1 antigen is a determinant of Stx binding to both erythrocytes and microvascular endothelial cells. If P and LKE blood group antigens also predict endothelial cell sensitivity to Stx injury, then P and LKE blood group phenotype may be clinically useful predictors of risk for development of HUS after STEC infection.

L9 ANSWER 6 OF 16 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 2001:232157 SCISEARCH
THE GENUINE ARTICLE: 410RA
TITLE: N-acetylgalactosamine, N-acetylglucosamine and sialic acid **expression** in primary breast cancers
AUTHOR: Brooks S A (Reprint); Carter T M
CORPORATE SOURCE: Oxford Brookes Univ, Res Sch Biol & Mol Sci, Gipsy Lane, Oxford OX3 0BP, England (Reprint); Oxford Brookes Univ, Res Sch Biol & Mol Sci, Oxford OX3 0BP, England
COUNTRY OF AUTHOR: England
SOURCE: ACTA HISTOCHEMICA, (FEB 2001) Vol. 103, No. 1, pp. 37-51. ISSN: 0065-1281.
PUBLISHER: URBAN & FISCHER VERLAG, BRANCH OFFICE JENA, P O BOX 100537, D-07705 JENA, GERMANY.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 42
ENTRY DATE: Entered STN: 30 Mar 2001
Last Updated on STN: 30 Mar 2001
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Binding of the lectin from Helix pomatia (HPA), which recognises N-acetylgalactosamine and N-acetylglucosamine glycans, is a predictor of metastasis and poor prognosis in a number of **human** adenocarcinomas, including breast cancer. The glycoproteins to which it binds in these tumours have been only partially characterised, and the

mechanisms underlying their biosynthesis remain unknown. In this study, 111 primary breast cancers were assessed for binding of HPA and labelling characteristics were compared directly with those of Dolichos biflorus agglutinin and soybean agglutinin, both of which also recognise N-acetylgalactosamine, Griffonia simplicifolia agglutinin II, which recognises N-acetylglucosamine, and Limax flavus agglutinin, Sambucus nigra agglutinin and Maackia amurensis lectin I, all of which recognise sialic acids. Results indicate that the HPA-binding partners **expressed** by cancer cells are predominantly N-acetylgalactosamine glycans, but some recognition of N-acetylglucosamine species is also likely. There was no evidence to support the hypothesis that overexpression of these moieties results from failure in sialylation. Alternative mechanisms, for example alterations in levels of activity of appropriate **glycosyl transferases** or disruption in transport and processing mechanisms leading to failure of normal chain extension of glycans may be responsible, and these are areas that warrant further investigation.

L9 ANSWER 7 OF 16 HCAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2000:193716 HCAPLUS

DOCUMENT NUMBER: 133:102935

TITLE: Altered mRNA **expression** of glycosyltransferases in human colorectal carcinomas and liver metastases

AUTHOR(S): Petretti, T.; Kemmner, W.; Schulze, B.; Schlag, P. M.

CORPORATE SOURCE: Department of Surgery and Surgical Oncology, Robert-Rossle-Klinik at the Max Delbrück Centre for Molecular Medicine, Berlin, 13125, Germany

SOURCE: Gut (2000), 46(3), 359-366

CODEN: GUTTAK; ISSN: 0017-5749

PUBLISHER: BMJ Publishing Group

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Biosynthesis of carbohydrate structures is tissue specific and developmentally regulated by **glycosyl-transferases** such as fucosyltransferases, sialyltransferases, and N-acetylglucosaminyltransferases. During carcinogenesis, aberrant glycosylation leads to the development of tumor subpopulations with different adhesion properties. Therefore alterations in glycosyltransferase mRNA **expression** in colorectal carcinomas were examined by semiquant. reverse transcription-polymerase chain reaction (RT-PCR). Colorectal carcinoma specimens were classified and characterized according to the WHO/UICC system. **Expression** of fucosyltransferases FT-I, FT-III, FT-IV, FT-V, FT-VI, and FT-VII, sialyltransferases ST3Gal-I, ST3Gal-III, ST3Gal-IV, and ST6Gal-I, β 1,4-galactosyltransferase, and β 1,6-Acetylglucosaminyltransferase V (GNT-V) was screened simultaneously in exts. of 22 homogenized tumor specimens by RT-PCR and compared with corresponding mucosa from each patient. Also 12 adenomas and 17 liver metastases of colorectal carcinomas were examined GNT-V **expression** was enhanced in colorectal adenomas ($p = 0.039$), carcinomas ($p < 0.001$), and liver metastases of colorectal carcinomas ($p < 0.001$). Also, **expression** of fucosyltransferase FT-IV was increased in colorectal adenomas ($p = 0.039$) and carcinomas ($p < 0.001$). In addition, fucosyltransferase FT-I ($p < 0.001$) and sialyltransferases ST6Gal-I ($p = 0.004$) and ST3Gal-III ($p = 0.001$) showed increased **expression** in carcinoma specimens. On the other hand, fucosyltransferase FT-III was less abundantly **expressed** in carcinomas exhibiting distant metastases ($p = 0.046$) and in highly invasive tumors ($p = 0.041$). Glycosyltransferase mRNA **expression** is significantly altered in colorectal adenomas and carcinomas isolated from surgical specimens. RT-PCR determination of specific glycosyltransferases may be helpful for earlier

detection of carcinomas and for tumor prognosis.

REFERENCE COUNT: 55 THERE ARE 55 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 8 OF 16 HCAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2000:178305 HCAPLUS

DOCUMENT NUMBER: 133:130435

TITLE: A new biological agent for treatment of Shiga
toxigenic Escherichia coli infections and dysentery in
humans

AUTHOR(S): Paton, Adrienne W.; Morona, Renato; Paton, James C.

CORPORATE SOURCE: Molecular Microbiology Unit, Women's and Children's
Hospital, North Adelaide, 5006, Australia

SOURCE: Nature Medicine (New York) (2000), 6(3), 265-270
CODEN: NAMEFI; ISSN: 1078-8956

PUBLISHER: Nature America

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Gastrointestinal disease caused by Shiga toxin-producing bacteria (such as
Escherichia coli O157:H7 and Shigella dysenteriae) is often complicated by
life-threatening toxin-induced systemic sequelae, including
hemolytic-uremic syndrome. Such infections can now be diagnosed very
early in the course of the disease, but at present no effective
therapeutic intervention is possible. Here, the authors constructed a
recombinant bacterium that displayed a Shiga toxin receptor mimic
on its surface, and it adsorbed and neutralized Shiga toxins with very
high efficiency. Moreover, oral administration of the **recombinant**
bacterium completely protected mice from challenge with an otherwise
100%-fatal dose of Shiga toxigenic E. coli. Thus, the bacterium shows
great promise as a "probiotic" treatment for Shiga toxigenic E. coli
infections and dysentery.

REFERENCE COUNT: 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 9 OF 16 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
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ACCESSION NUMBER: 2000197399 EMBASE

TITLE: Role of oligosaccharides and glycoconjugates in intestinal
host defense.

AUTHOR: Dai D.; Nanthkumar N.N.; Newburg D.S.; Walker W.A.

CORPORATE SOURCE: Dr. W.A. Walker, Division of Nutrition, Harvard Medical
School, C. Hosp./Massachusetts General Hosp., 300 Longwood
Avenue, Boston, MA 02115, United States

SOURCE: Journal of Pediatric Gastroenterology and Nutrition, (2000)
Vol. 30, No. SUPPL. 2, pp. S23-S33.

Refs: 96

ISSN: 0277-2116 CODEN: JPGND6

COUNTRY: United States

DOCUMENT TYPE: Journal; Conference Article

FILE SEGMENT: 004 Microbiology

007 Pediatrics and Pediatric Surgery

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 20000630

Last Updated on STN: 20000630

AB The attachment of microbes to carbohydrates moieties on the host cell
surface is considered essential for successful colonization and infection.
Adding sugars to MVM membrane proteins and lipids (glycosylation) is an
important host determinant in microbial colonization of the
intestine. The enzymes responsible for glycosylation are
glycosyltransferases. Our studies and others have shown that
glycosyltransferases and microbial receptors are under developmental
regulation. Intrinsic genetic control, hormones (glucocorticoids, insulin

and thyroxine), and external factors (diet and bacterial colonization) may affect the ontogeny of these enzymes and the **expression** of microbial receptors. Therefore, the developmental control of microbial receptors in the gastrointestinal tract may in part contribute to the altered host sensitivity to intestinal infection in infancy. Probiotics and some trophic factors may also protect the gastrointestinal tract through differential glycosylation. In the future, it may also be possible to inhibit microbial attachment by blocking with oligosaccharides or glycoconjugates specific for the appropriate lectins. The molecular nature of microbial receptors in intestinal epithelial cells underscores the importance of intestinal surface carbohydrate **expression** in host-microbe interaction. With improved techniques for characterizing receptor binding and the receptor's structure - i.e., the availability of several **human** intestinal models (organ culture of **human** fetus **intestine**, primary culture of **human** fetus intestinal epithelial cells, the H-4 cell line, and Caco-2 cell line) and of carbohydrate-specific monoclonal antibodies, we may identify additional membrane receptors and the receptor sugar sequences in the near future. We may isolate glycoconjugates from **human** intestinal tissue, then identify them structurally using mass spectrometry and nuclear magnetic resonance spectroscopy. We may test the binding of microbial ligands to epithelial surfaces with glycoproteins or glycolipids. Subsequent studies on the intestinal **expression** and developmental regulation of individual glycosyltransferases can then be pursued. Recently, a transgenic mouse model has been used to study *Helicobacter pylori* infection, in which the receptor, the primate-specific Lewisb (95), was **expressed** in the mouse gastrointestinal tract by transfection with a **human** α -1,3/4- fucosyltransferase (96). In the future, the potential transgenic animal models by transfection with constructs for specific **glycosyl-transferase(s)** will be used to examine the role of oligosaccharides and glycoconjugates in regulating cellular differentiation and the host-microbe interaction. In the same manner, the use of molecular and cell biologic techniques in intestinal cell lines and in primary cultures of **human** enterocytes or organ culture of **human** fetal, neonatal, and adult **intestine** will help to determine the relationship between developmental regulation of intestinal microbial receptors and postreceptor-effector events. By understanding the molecular nature of microbial receptors and their effector responses in the **intestine**, the developmental programming and environmental influence on receptor **expression**, and the effector response and the biologic significance in neonatal host defenses, new approaches may soon be available in the prevention and treatment of infants with infectious intestinal diseases of various origins.

L9 ANSWER 10 OF 16 HCAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 1999:691109 HCAPLUS

DOCUMENT NUMBER: 131:335805

TITLE: Glycosylation engineering of antibodies for improving antibody-dependent cellular cytotoxicity

INVENTOR(S): Umana, Pablo; Jean-Mairet, Joel; Bailey, James E.

PATENT ASSIGNEE(S): Switz.

SOURCE: PCT Int. Appl., 79 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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WO 9954342	A1	19991028	WO 1999-US8711	19990420
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ,				

DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

AU 9936578	A1	19991108	AU 1999-36578	19990420
EP 1071700	A1	20010131	EP 1999-918731	19990420

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI

JP 2002512014	T2	20020423	JP 2000-544680	19990420
US 6602684	B1	20030805	US 1999-294584	19990420
US 2004072290	A1	20040415	US 2003-437388	20030514
US 2005074843	A1	20050407	US 2003-633699	20030805
US 2005079605	A1	20050414	US 2003-633697	20030805

PRIORITY APPLN. INFO.: US 1998-82581P P 19980420
US 1999-294584 A1 19990420
WO 1999-US8711 W 19990420

AB The present invention relates to the field of glycosylation engineering of proteins. More particularly, the present invention is directed to the glycosylation engineering of proteins to provide proteins with improved therapeutic properties, e.g., antibodies, antibody fragments, or a fusion protein that includes a region equivalent to the Fc region of an Ig, with enhanced Fc-mediated cellular cytotoxicity. The antibodies or fusion proteins with enhanced Fc-mediated cellular cytotoxicity are **expressed** in host cells engineered to also **express** a glycoprotein-modifying **glycosyl transferase**, e.g. $\beta(1,4)$ -N-acetylglucosaminyltransferase III or V, $\beta(1,4)$ -N-galactosyltransferase, and mannosidase II.

REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 11 OF 16 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN

ACCESSION NUMBER: 1995:510717 BIOSIS
DOCUMENT NUMBER: PREV199598515767
TITLE: **Expression** and regulation of glycosyltransferases for N-glycosyl oligosaccharides in fresh **human** surgical and **murine** tissues and cultured cell lines.

AUTHOR(S): Li, M.; Andersen, V.; Lance, P. [Reprint author]
CORPORATE SOURCE: Div. Gastroenterol., Buffalo General Hosp., 100 High St., Buffalo, NY 14203, USA
SOURCE: Clinical Science (London), (1995) Vol. 89, No. 4, pp. 397-404.
CODEN: CSCIAE. ISSN: 0143-5221.

DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 29 Nov 1995
Last Updated on STN: 27 Jan 1996

AB 1. Mammalian membrane and serum proteins are glycosylated by the addition of heterogeneous N-linked oligosaccharides. It has been widely speculated that oligosaccharide diversity is achieved by corresponding heterogeneity of **expression** of the **glycosyl-transferases** that are responsible for oligosaccharide synthesis. 2. We surveyed mRNA levels of three sequentially acting glycosyltransferases, N-acetylglucosaminyltransferase 1, beta-1,4-galactosyltransferase and alpha-2,6-sialyltransferase, in 11 **human** tissues and confirmed the expected variations. 3. The size heterogeneity of alpha-2,6-sialyltransferase transcripts reported in rat tissues was evident neither in the **human** tissue survey nor in a panel of

murine RNAs. Tissue distributions of alternative terminal sialyltransferases, alpha-2,6-sialyltransferase and alpha-2,3-sialyltransferase, were distinct. 4. Relative glycosyltransferase mRNA levels in four transformed **human** cell lines cultured in vitro did not fully reflect levels in the corresponding **human** tissues. 5. **Expression** of alpha-2,6-sialyltransferase mRNA was approximately 2.6-fold greater in adenocarcinomatons than in normal **human colon**, and beta-1,4-galactosyltransferase **expression** was approximately 1.8-fold greater in normal than in adenocarcinomatons **colon**. 6. n-Butyrate (0.003-0.005mol/l), a short-chain fatty acid that is produced by colonic bacterial fermentation, caused approximately 80% inhibition of alpha-2,6-sialyltransferase, approximately 2.5-fold induction of beta-1,4-galactosyltransferase and approximately 6-fold induction of N-acetylglucosaminyltransferase mRNAs in T84 (colonic) cells. The effects on alpha-2,6sialyltransferase and beta-1,4-galactosyltransferase were near maximal by 6 h, but induction of N-acetylglucosaminyltransferase was fully apparent only after exposure for 24 h.

L9 ANSWER 12 OF 16 MEDLINE on STN DUPLICATE 2
 ACCESSION NUMBER: 95013396 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 7928404
 TITLE: Glycoconjugates of the normal **human** colorectum: a lectin histochemical study.
 AUTHOR: McMahon R F; Panesar M J; Stoddart R W
 CORPORATE SOURCE: Department of Pathological Sciences, University of Manchester, UK.
 SOURCE: Histochemical journal, (1994 Jun) 26 (6) 504-18.
 Journal code: 0163161. ISSN: 0018-2214.
 PUB. COUNTRY: ENGLAND: United Kingdom
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 199411
 ENTRY DATE: Entered STN: 19941222
 Last Updated on STN: 19941222
 Entered Medline: 19941108

AB Previous studies of the normal **human** colorectum by lectin histochemistry have used a mixture of tissues, including those derived from **colons** harbouring neoplasia and inflammatory bowel diseases. In the current investigation, tissues from patients without either of these conditions have been examined with a wide panel of lectins, encompassing specificities directed against both N- and O-linked sequences, using an avidin peroxidase revealing system and evaluated with a semiquantitative scoring method. The results of binding of these lectins have been compared with those seen in the resection margins of (at least 5 cm away from) colorectal carcinomas. Consistent regional variations were noted between right- and left-sided colonic tissues, with more diverse glycan structures and a greater sialyl content in the distal **colon**. There was evidence of graduation of formation of oligosaccharide chains in developing crypts, possibly related to the maturation and **expression** of **glycosyl transferases** responsible for the incorporation of mannose residues of N-linked oligosaccharides and of N-acetylgalactosamine and N-acetylglucosamine. Comparison with previous reports has revealed some variations, possibly related to tissue fixation and processing and to lectin concentrations employed, which raises the question of standardization of methodologies in lectin histochemical investigations.

L9 ANSWER 13 OF 16 HCAPLUS COPYRIGHT 2005 ACS on STN
 ACCESSION NUMBER: 1994:51402 HCAPLUS
 DOCUMENT NUMBER: 120:51402
 TITLE: Biosynthetic basis of incompatible histo-blood group A

antigen **expression**: Anti-A transferase
 antibodies reactive with gastric **cancer**
tissue of type O individuals
 AUTHOR(S): David, Leonor; Leitao, Dina; Sobrinho-Simoes, Manuel;
 Bennett, Eric Paul; White, Thayer; Mandel, Ulla;
 Dabelsteen, Erik; Clausen, Henrik
 CORPORATE SOURCE: Fac. Med., Univ. Porto, Porto, 4200, Port.
 SOURCE: Cancer Research (1993), 53(22), 5494-500
 CODEN: CNREA8; ISSN: 0008-5472

DOCUMENT TYPE: Journal
 LANGUAGE: English

AB The **expression** of incompatible A carbohydrate antigens in some adenocarcinomas may provide an explanation for the generally observed lower incidence of adenocarcinoma among types O and B vs. type A individuals. The chemical and genetic basis of incompatible A **expression** is largely unknown. In this study, the authors have screened 31 cases of gastric tumors of phenotype O for the **expression** of blood group A gene-defined glycosyltransferase by immunohistol. on frozen sections using newly developed monoclonal antibodies to the transferases. Three cases were pos., and transferase **expression** was confirmed by enzyme anal. of exts. from the specimens. Blood group A carbohydrate antigens were also identified immunohistol. in these three cases as well as in five other cases. Thin-layer chromatog. immunostaining anal. of glycolipid exts. from the three cases did not confirm the chemical presence of A antigen. The ABO genotype of all patients was found to be OO, showing that all carried O alleles with a structural defect at nucleotide position 261 leading to a shift in the reading frame. The data suggest that incompatible A antigen **expression** is a result of transferase **expression** derived from the ABO genes.

L9 ANSWER 14 OF 16 HCAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 1991:600344 HCAPLUS
 DOCUMENT NUMBER: 115:200344
 TITLE: **Cloning** of cDNA for **human**
 intestinal mucins
 INVENTOR(S): Kim, Young S.; Gum, James R., Jr.
 PATENT ASSIGNEE(S): University of California, Oakland, USA
 SOURCE: PCT Int. Appl., 57 pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9108217	A1	19910613	WO 1990-US7087	19901204
W: CA, JP				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, NL, SE				
PRIORITY APPLN. INFO.:			US 1989-447140	A 19891205

AB The cDNA for **human** intestinal mucins is **cloned**. The cDNA may be **expressed** in **recombinant** cells and antibodies to the nonglycosylated mucin (e.g. that obtained by **expressing** the cDNA in a prokaryote) may be used to diagnose diseases such as epithelial cancer. **Human colon** cancer intestinal mucins were isolated and chemical deglycosylatd. Antisera to these proteins was used to screen a cDNA library from **human intestine**. Several **clones** were isolated and sequenced. All contained tandem repeats of 69 nucleotides encoding a threonine- and proline-rich peptide. In vitro translation of poly A RNA from **human small intestine, colon, and colon** cancer cells produced a 162,000-dalton protein which was immunopptd. with antibodies to deglycosylatd mucin. One of the cDNAs was

used in Southern blotting expts. This indicated the presence of restriction fragment length polymorphisms in the intestinal mucin gene. Other **human** intestinal mucin cDNAs were isolated which belonged to a 2nd class of mucins. These contained HSTPSFTSSITTTETTS repeating units.

L9 ANSWER 15 OF 16 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
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ACCESSION NUMBER: 91103617 EMBASE
DOCUMENT NUMBER: 1991103617
TITLE: Regulation of the oncodevelopmental **expression** of type 1 chain ABH and Lewisb blood group antigens in **human colon** by α -2-L-fucosylation.
AUTHOR: Orntoft T.F.; Greenwell P.; Clausen H.; Watkins W.M.
CORPORATE SOURCE: Department of Experimental, Clinical Oncology, Danish Cancer Society, Norrebrogade 44, DK-8000 Aarhus C, Denmark
SOURCE: Gut, (1991) Vol. 32, No. 3, pp. 287-293.
ISSN: 0017-5749 CODEN: GUTTAK
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 016 Cancer
025 Hematology
029 Clinical Biochemistry
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 911216
Last Updated on STN: 911216

AB Blood group antigen **expression** in the distal **human colon** is related to the development of the organ and is modified by malignant transformation. To elucidate the biochemical basis for these changes, we have (a) analysed the activity of **glycosyl transferases** coded for by the H, Se, Le, X, and A genes, in tissue biopsy specimens from normal and malignant proximal and distal **human colon**; (b) characterised the glycosphingolipids **expressed** in the various regions of normal and malignant **colon** by immunostaining of high performance thin layer chromatography plates; and (c) located the antigens on tissue sections from the same subjects by immunohistochemistry. In both secretors and non-secretors we found a significantly higher activity of α -2-L-fucosyltransferases in carcinomatous rectal tissue than in tissue from normal subjects, whereas the other transferase activities studied showed no significant differences. The acceptor substrate specificity suggested that both the Se and the H gene dependent α -2-L-fucosyltransferases are increased in carcinomas. In non-malignant tissue the only enzyme which showed appreciably higher activity in caecum than in rectum was α -2-L-fucosyltransferase. Immunochemistry and immunohistochemistry showed α -2-L-fucosylated structures in normal caecum from secretors and in tumour tissue from both secretors and non-secretors. We conclude that the α -2-L-fucosyltransferases control the **expression** of ABH, and Lewis(b) structures in normal and malignant **colon**.

L9 ANSWER 16 OF 16 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN

ACCESSION NUMBER: 88161953 EMBASE
DOCUMENT NUMBER: 1988161953
TITLE: Lectin **expression** in neoplastic and non-neoplastic lesions of the rectum.
AUTHOR: Lee Y.-S.
CORPORATE SOURCE: Department of Pathology, National University of Singapore, Singapore 0511, Singapore
SOURCE: Pathology, (1988) Vol. 20, No. 2, pp. 157-165.

ISSN: 0031-3205 CODEN: PTLGAX
 COUNTRY: Australia
 DOCUMENT TYPE: Journal
 FILE SEGMENT: 005 General Pathology and Pathological Anatomy
 016 Cancer
 048 Gastroenterology
 LANGUAGE: English
 SUMMARY LANGUAGE: English
 ENTRY DATE: Entered STN: 911211
 Last Updated on STN: 911211

AB The **expression** of six lectins (Arachis hypogaea, B. simplicifolia I, concanavalin A, Dolichus biflorus, Triticum vulgare, Lotus tetragonolobus) was studied in 24 adenocarcinomas, 24 adenomas, 20 metaplastic polyps, 17 specimens of mucosal prolapse (solitary ulcer syndrome) and 10 of normal mucosa, all taken from the rectum. Qualitative, quantitative and distributive differences in lectin **expression** were observed between adenocarcinoma and normal mucosa. These cancer-associated glycoprotein alterations were also observed, though to a lesser extent, in benign neoplastic and non-neoplastic lesions of the rectum. It appears therefore that the glycoprotein modifications associated with malignant transformation are not specific indicators of malignancy. It is suggested that the common denominator is a disturbance in the activities of enzymes, particularly the **glycosyl-transferases** and glycosidases, involved in the biosynthesis of glycoprotein. This disturbance can occur in situations where cells are less differentiated either through developmental immaturity, rapid cellular division or neoplastic de-differentiation. These changes are therefore more likely to reflect the state of differentiation rather than the malignant nature of the cells. It is shown that the greater the deviation of the lesion from normal the greater the glycoprotein alterations. The potential usefulness of lectin **expressions** as predictive indicators of biological behaviour of adenocarcinomas of the large bowel needs further studies.

=> d his

(FILE 'HOME' ENTERED AT 08:53:43 ON 29 JUL 2005)

FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS, LIFESCI' ENTERED AT 08:54:06 ON 29 JUL 2005

L1 2838 S GLYCOSYL (A)TRANSFERASE?
 L2 594 S (HUMAN OR MURINE) AND L1
 L3 3 S "GST4ALPHA"
 L4 1 DUP REM L3 (2 DUPLICATES REMOVED)
 L5 7189603 S CLON? OR EXPRESS? OR RECOMBINANT
 L6 213 S L2 AND L5
 L7 1025751 S COLON OR INTESTINE OR (CANCER(A)TISSUE?)
 L8 21 S L6 AND L7
 L9 16 DUP REM L8 (5 DUPLICATES REMOVED)

=> s transmembrane

L10 258785 TRANSMEMBRANE

=> s 16 and 110

L11 8 L6 AND L10

=> dup rem 111

PROCESSING COMPLETED FOR L11

L12 7 DUP REM L11 (1 DUPLICATE REMOVED)

=> d 1-7 ibib ab

L12 ANSWER 1 OF 7 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
 ACCESSION NUMBER: 2004:320923 BIOSIS
 DOCUMENT NUMBER: PREV200400322177
 TITLE: Functional characterization of a novel *Toxoplasma gondii* glycosyltransferase: UDP-N-acetyl-D-galactosamine:polypeptide N-acetylgalactosaminyltransferase-T3.
 AUTHOR(S): Stwora-Wojczyk, Magdalena M.; Dzierszynski, Florence; Roos, David S.; Spitalnik, Steven L.; Wojczyk, Boguslaw S.
 [Reprint Author]
 CORPORATE SOURCE: Ctr MedDept Pathol and Lab Med, Univ Rochester, Box 626,601 Elmwood Ave, Rochester, NY, 14642, USA
 boguslaw_wojczyk@urmc.rochester.edu
 SOURCE: Archives of Biochemistry and Biophysics, (June 15 2004)
 Vol. 426, No. 2, pp. 231-240. print.
 ISSN: 0003-9861 (ISSN print).
 DOCUMENT TYPE: Article
 LANGUAGE: English
 ENTRY DATE: Entered STN: 21 Jul 2004
 Last Updated on STN: 21 Jul 2004

AB We report the functional characterization of a new UDP-GalNAc:polypeptide N-acetylgalactosaminyltransferase (ppGalNAc-T) (EC 2.4.1.41) from the **human** disease-causing parasite, *Toxoplasma gondii*. This glycosyltransferase is denoted as T *gondii* ppGalNAc-T3. These enzymes are responsible for the initial step of mucin-type O-glycosylation: the transfer of GalNAc from the UDP-GalNAc nucleotide shear donor onto a peptide acceptor. Following an in silico analysis of the publicly available T *gondii* DNA database, we used molecular biology approaches to identify and isolate the cDNA encoding this enzyme. The resulting type II membrane protein contains N-terminal cytoplasmic, **transmembrane**, and C-terminal lumenal domains. Conceptual translation of the cDNA sequence also reveals a stein region and the presence of several important sequence motifs. When the **recombinant** construct was **expressed** in stably transfected *Drosophila melanogaster* S2 cells, the purified protein exhibited glycosyltransferase activity in vitro against glycopeptide, but not "naked" peptide, acceptors. In addition, using reverse transcriptase-PCR, T. *gondii* ppGalNAc-T3 mRNA was equivalently **expressed** during the tachyzoite and bradyzoite developmental stages. The identification of T *gondii* ppGalNAc-T3 as a functional "follow-up" glycopeptide glycosyltransferase further confirms that this **human** parasite has its own enzymatic O-glycosylation machinery. Copyright 2004 Elsevier Inc. All rights reserved.

L12 ANSWER 2 OF 7 HCAPLUS COPYRIGHT 2005 ACS on STN
 ACCESSION NUMBER: 2003:379629 HCAPLUS
 DOCUMENT NUMBER: 139:66640
 TITLE: Transcription starting from an alternative promoter leads to the **expression** of the **human** ABO histo-blood group antigen
 AUTHOR(S): Hata, Yukiko; Kominato, Yoshihiko; Takizawa, Hisao; Tabata, Sachiyo; Michino, Junko; Nishino, Kazuma; Yasumura, Satoshi; Yamamoto, Fumiichiro
 CORPORATE SOURCE: Faculty of Medicine, Department of Legal Medicine, Toyama Medical and Pharmaceutical University, Japan
 SOURCE: Transfusion (Malden, MA, United States) (2003), 43(5), 656-662
 CODEN: TRANAT; ISSN: 0041-1132
 PUBLISHER: Blackwell Publishing, Inc.
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 AB Using the 5'-rapid amplification of cDNA ends technique with the ex vivo culture of AC133-CD34+ cells, a transcription start site was recently identified approx. 0.7 kb upstream from the transcription start sites

previously determined. The transcripts from the alternative starting exon 1a were demonstrated in the cells of both erythroid and epithelial lineages. Because the nucleotide sequence of exon 1a does not contain an ATG codon, we examined whether transcription starting from exon 1a leads to production of a

functional **glycosyl-transferase**. Stable transfection expts. into the **human** gastric cancer MKN28 cells were performed using the various A transferase **expression** plasmids. Large amts. of A antigens were demonstrated on the cells transfected with the A transferase **expression** plasmid containing the entire cDNA from exon 1a or the 5'-truncated cDNA leading to the production of the N-truncated protein with deletion of the cytoplasmic tail and a portion of the **transmembrane** domain. However, negligible amts. of A antigens were observed on the cells transfected with the A transferase **expression** plasmids containing the 5'-truncated cDNA leading to the production of the N-truncated proteins without the cytoplasmic tail and the **transmembrane** domain. This study suggests that a functional A transferase could be produced by the transcription from exon 1a.

REFERENCE COUNT: 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 3 OF 7 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 2002:433092 BIOSIS
DOCUMENT NUMBER: PREV200200433092
TITLE: Demonstration of a novel gene DEXT3 of Drosophila melanogaster as the essential N-acetylglucosamine transferase in the heparan sulfate biosynthesis. Chain initiation and elongation.
AUTHOR(S): Kim, Byung-Taek; Kitagawa, Hiroshi; Tamura, Jun-ichi; Kusche-Gullberg, Marion; Lindahl, Ulf; Sugahara, Kazuyuki [Reprint author]
CORPORATE SOURCE: Dept. of Biochemistry, Kobe Pharmaceutical University, 4-19-1 Motoyamakita-machi, Higashinada-ku, Kobe, 658-8558, Japan
k-sugar@kobepharm-u.ac.jp
SOURCE: Journal of Biological Chemistry, (April 19, 2002) Vol. 277, No. 16, pp. 13659-13665. print.
CODEN: JBCHA3. ISSN: 0021-9258.
DOCUMENT TYPE: Article
LANGUAGE: English
OTHER SOURCE: Genbank-AB077850; DDBJ-AB077850; Genbank-AF132161
ENTRY DATE: Entered STN: 14 Aug 2002
Last Updated on STN: 23 Sep 2002

AB Hereditary multiple exostoses gene (EXT) family members encode glycosyltransferases required for heparan sulfate (HS) biosynthesis in **humans** as well as in Drosophila. In the present study, we identified a novel Drosophila EXT protein with a type II **transmembrane** topology and demonstrated its glycosyltransferase activities. The truncated soluble form of this new homolog designated DEXT3 transferred N-acetylglucosamine (GlcNAc) through an alpha1,4-linkage not only to N-acetylheparosan oligosaccharides that represent growing HS chains (alpha-GlcNAc transferase II activity) but also to GlcUAbeta1-3Galbeta1-O-C2H4NHCBz, a synthetic substrate for alpha-GlcNAc transferase I that determines and initiates HS biosynthesis. The results suggest that DEXT3 is the ortholog of **human** EXTL3 and Caenorhabditis elegans rib-2. Semiquantitative reverse transcriptase-PCR analysis revealed ubiquitous **expression** of the DEXT3 mRNA. Based on the findings of the present study and those of a recent study where a fly mutant, deficient in the botv gene identical to DEXT3, affected HS proteoglycan-mediated developmental signalings, it is suggested that DEXT3 with the revealed **glycosyl-transferase** activities is critically involved in HS formation in Drosophila. These results suggest the essential roles of DEXT3, its

human ortholog EXTL3, and the C. elegans ortholog rib-2 in the biosynthesis of heparan sulfate and heparin, if present, in the respective organisms.

L12 ANSWER 4 OF 7 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation on STN

ACCESSION NUMBER: 2002:905924 SCISEARCH

THE GENUINE ARTICLE: 609WZ

TITLE: **Human** airway mucin glycosylation: A combinatorial of carbohydrate determinants which vary in cystic fibrosis
AUTHOR: Lamblin G; Degroote S; Perini J M; Delmotte P; Scharfman A E; Davril M; Lo-Guidice J M; Houdret N; Dumur V; Klein A; Roussel P (Reprint)

CORPORATE SOURCE: Fac Med, INSERM, U 377, Dept Biochim, Pl Verdun, F-59045 Lille, France (Reprint); Fac Med, INSERM, U 377, Dept Biochim, F-59045 Lille, France; Univ Lille 2, F-59045 Lille, France

COUNTRY OF AUTHOR: France

SOURCE: GLYCOCONJUGATE JOURNAL, (SEP 2001) Vol. 18, No. 9, pp. 661-684.

ISSN: 0282-0080.

PUBLISHER: KLUWER ACADEMIC PUBL, VAN GODEWIJCKSTRAAT 30, 3311 GZ DORDRECHT, NETHERLANDS.

DOCUMENT TYPE: General Review; Journal

LANGUAGE: English

REFERENCE COUNT: 225

ENTRY DATE: Entered STN: 26 Nov 2002

Last Updated on STN: 26 Nov 2002

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB **Human** airway mucins represent a very broad family of polydisperse high molecular mass glycoproteins, which are part of the airway innate immunity. Apomucins, which correspond to their peptide part, are encoded by at least 6 different mucin genes (MUC1, MUC2, MUC4, MUC5B, MUC5AC and MUC7). The **expression** of some of these genes (at least MUC2 and MUC5AC) is induced by bacterial products, tobacco smoke and different cytokines.

Human airway mucins are highly glycosylated (70-80% per weight). They contain from one single to several hundred carbohydrate chains. The carbohydrate chains that cover the apomucins are extremely diverse, adding to the complexity of these molecules. Structural information is available for more than 150 different O-glycan chains corresponding to the shortest chains (less than 12 sugars).

The biosynthesis of these carbohydrate chains is a stepwise process involving many glycosyl- or sulfo-transferases. The only structural element shared by all mucin O-glycan chains is a GalNAc residue linked to a serine or threonine residue of the apomucin. There is growing evidence that the apomucin sequences influence the first glycosylation reactions. The elongation of the chains leads to various linear or branched extensions. Their non-reducing end, which corresponds to the termination of the chains, may bear different carbohydrate structures, such as histo-blood groups A or B determinants, H and sulfated H determinants, Lewis a, Lewis b, Lewis x or Lewis y epitopes, as well as sialyl- or sulfo- (sometimes sialyl- and sulfo-) Lewis a or Lewis x determinants. The synthesis of these different terminal determinants involves three different pathways with a whole set of glycosyl- and sulfo- transferases.

Due to their wide structural diversity forming a combinatorial of carbohydrate determinants as well as their location at the surface of the airways, mucins are involved in multiple interactions with microorganisms and are very important in the protection of the underlying airway mucosa.

Airway mucins are oversulfated in cystic fibrosis and this feature has been considered as being linked to a primary defect of the disease. However, a similar pattern is observed in mucins from patients suffering from chronic bronchitis when they are severely infected. Airway mucins

from severely infected patients suffering either from cystic fibrosis or from chronic bronchitis are also highly sialylated, and highly **express** sialylated and sulfated Lewis x determinants, a feature which may reflect severe mucosal inflammation or infection.

These determinants are potential sites of attachment for *Pseudomonas aeruginosa*, the pathogen responsible for most of the morbidity and mortality in cystic fibrosis, and the **expression** of the sulfo- and **glycosyl-transferases** involved in their biosynthesis is increased by TNF α .

In summary, airway inflammation may simultaneously induce the **expression** of mucin genes (MUC2 and MUC5AC) and the **expression** of several glycosyl- and sulfo- transferases, therefore modifying the combinatory glycosylation of these molecules.

L12 ANSWER 5 OF 7 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation on STN

ACCESSION NUMBER: 2000:5881 SCISEARCH

THE GENUINE ARTICLE: 266AV

TITLE: The cytoplasmic, **transmembrane**, and stem regions of glycosyltransferases specify their in vivo functional sublocalization and stability in the Golgi

AUTHOR: Grabenhorst E (Reprint); Conradt H S

CORPORATE SOURCE: Gesell Biotechnol Forsch GmbH, Prot Glycosylat Grp, Mascheroder Weg 1, D-38124 Braunschweig, Germany (Reprint); Gesell Biotechnol Forsch GmbH, Prot Glycosylat Grp, D-38124 Braunschweig, Germany

COUNTRY OF AUTHOR: Germany

SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (17 DEC 1999) Vol. 274, No. 51, pp. 36107-36116.

ISSN: 0021-9258.

PUBLISHER: AMER SOC BIOCHEMISTRY MOLECULAR BIOLOGY INC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814 USA.

DOCUMENT TYPE: Article; Journal

LANGUAGE: English

REFERENCE COUNT: 68

ENTRY DATE: Entered STN: 2000

Last Updated on STN: 2000

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB We provide evidence for the presence of targeting signals in the cytoplasmic, **transmembrane**, and stem (CTS) regions of Golgi glycosyltransferases that mediate sorting of their intracellular catalytic activity into different functional subcompartmental areas of the Golgi. We have constructed chimeras of **human** alpha 1,3-fucosyltransferase VI (FT6) by replacement of its CTS region with those of late and early acting Golgi glycosyltransferases and have stably coexpressed these constructs in BHK-81 cells together with the secretory reporter glycoprotein **human** beta-trace protein. The sialyl Lewis X:Lewis X ratios detected in beta-trace protein indicate that the CTS regions of the early acting GlcNAc-transferases I (GnT-I) and III (GnT-III) specify backward targeting of the FT6 catalytic domain, whereas the CTS region of the late acting **human** alpha 1,3-fucosyltransferase VII (FT7) causes forward targeting of the FT6 in vivo activity in the biosynthetic glycosylation pathway. The analysis of the in vivo functional activity of nine different CTS chimeras toward beta-trace protein allowed for a mapping of the CTS donor glycosyltransferases within the Golgi/trans-Golgi network: GnT-I < (ST6Gal I, ST3Gal III) < GnT-III < ST8Sia IV < GalT-I < (FT3, FT6) < ST3Gal TV < FT7. The sensitivity or resistance of the donor **glycosyl transferases** toward intracellular proteolysis is transferred to the chimeric enzymes together with their CTS regions. Apparently, there are at least three different signals contained in the CTS regions of glycosyltransferases mediating: first, their Golgi retention; second, their targeting to specific in vivo functional areas; and third, their susceptibility toward intracellular proteolysis as a tool

for the regulation of the intracellular turnover.

L12 ANSWER 6 OF 7 HCAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 1992:35636 HCAPLUS
DOCUMENT NUMBER: 116:35636
TITLE: **Cloning and expression** of DNA for
mammalian glycosidating enzymes
INVENTOR(S): Lowe, John B.
PATENT ASSIGNEE(S): University of Michigan, USA
SOURCE: PCT Int. Appl., 159 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9112340	A1	19910822	WO 1991-US899	19910214
W: CA, JP, KR				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, NL, SE				
CA 2075949	AA	19910815	CA 1991-2075949	19910214
EP 515536	A1	19921202	EP 1991-905027	19910214
EP 515536	B1	20000119		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE				
JP 05504480	T2	19930715	JP 1991-504707	19910214
AT 188999	E	20000215	AT 1991-905027	19910214
PRIORITY APPLN. INFO.:			US 1990-479858	A 19900214
			US 1990-480133	A 19900214
			US 1990-627621	A 19901212
			WO 1991-US899	W 19910214

AB A method of **cloning** genes or cDNAs for glycosyltransferase by transient **expression** in animal cells is described. The **cloned** sequences are useful in the study of the biol., pathol. and etiol. of glucosyl transferase-associated disease (no data). A cDNA encoding
a β -D-galactosyl-1,4-N-acetyl-D-glucosamide- α -1,3-galactosyltransferase (α 1-3 GT) was **cloned** from a bank in pCDM7 by screening transfected COS cells for binding to the lectin GS I-B4 introduced by glycosidation of cell surface polylactosamines. The cDNA sequence indicates that the enzymes may be a **transmembrane** protein with similarities to other glycosyltransferase.

L12 ANSWER 7 OF 7

MEDLINE on STN

DUPLICATE 1

ACCESSION NUMBER: 92098565 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1757461
TITLE: Isolation, characterization, and **expression** of cDNAs encoding **murine** alpha-mannosidase II, a Golgi enzyme that controls conversion of high mannose to complex N-glycans.
AUTHOR: Moremen K W; Robbins P W
CORPORATE SOURCE: Center for Cancer Research, Massachusetts Institute of Technology, Cambridge 02139.
CONTRACT NUMBER: CA14051 (NCI)
CA26712 (NCI)
GM31318 (NIGMS)
+
SOURCE: Journal of cell biology, (1991 Dec) 115 (6) 1521-34.
Journal code: 0375356. ISSN: 0021-9525.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals

OTHER SOURCE: GENBANK-M60692; GENBANK-M60693; GENBANK-M60694;
GENBANK-M60695; GENBANK-M60696; GENBANK-M60697;
GENBANK-S66758; GENBANK-S66760; GENBANK-S66765;
GENBANK-S73487; GENBANK-X61172

ENTRY MONTH: 199202

ENTRY DATE: Entered STN: 19920223
Last Updated on STN: 19960129
Entered Medline: 19920205

AB Golgi alpha-mannosidase II (GlcNAc transferase I-dependent alpha 1,3[alpha 1,6] mannosidase, EC 3.2.1.114) catalyzes the final hydrolytic step in the N-glycan maturation pathway acting as the committed step in the conversion of high mannose to complex type structures. We have isolated overlapping **clones** from a **murine** cDNA library encoding the full length alpha-mannosidase II open reading frame and most of the 5' and 3' untranslated region. The coding sequence predicts a type II **transmembrane** protein with a short cytoplasmic tail (five amino acids), a single **transmembrane** domain (21 amino acids), and a large COOH-terminal catalytic domain (1,124 amino acids). This domain organization which is shared with the Golgi **glycosyl-transferases** suggests that the common structural motifs may have a functional role in Golgi enzyme function or localization. Three sets of polyadenylated **clones** were isolated extending 3' beyond the open reading frame by as much as 2,543 bp. Northern blots suggest that these polyadenylated **clones** totaling 6.1 kb in length correspond to minor message species smaller than the full length message. The largest and predominant message on Northern blots (7.5 kb) presumably extends another approximately 1.4-kb downstream beyond the longest of the isolated **clones**. Transient **expression** of the alpha-mannosidase II cDNA in COS cells resulted in 8-12-fold overexpression of enzyme activity, and the appearance of cross-reactive material in a perinuclear membrane array consistent with a Golgi localization. A region within the catalytic domain of the alpha-mannosidase II open reading frame bears a strong similarity to a corresponding sequence in the rat liver endoplasmic reticulum alpha-mannosidase and the vacuolar alpha-mannosidase of *Saccharomyces cerevisiae*. Partial **human** alpha-mannosidase II cDNA **clones** were also isolated and the gene was localized to **human** chromosome 5.

=> d his

(FILE 'HOME' ENTERED AT 08:53:43 ON 29 JUL 2005)

FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS, LIFESCI' ENTERED AT 08:54:06 ON 29 JUL 2005

L1 2838 S GLYCOSYL (A)TRANSFERASE?
L2 594 S (HUMAN OR MURINE) AND L1
L3 3 S "GST4ALPHA"
L4 1 DUP REM L3 (2 DUPLICATES REMOVED)
L5 7189603 S CLON? OR EXPRESS? OR RECOMBINANT
L6 213 S L2 AND L5
L7 1025751 S COLON OR INTESTINE OR (CANCER(A)TISSUE?)
L8 21 S L6 AND L7
L9 16 DUP REM L8 (5 DUPLICATES REMOVED)
L10 258785 S TRANSMEMBRANE
L11 8 S L6 AND L10
L12 7 DUP REM L11 (1 DUPLICATE REMOVED)

=> e rosen s d/au

E1 8 ROSEN S B/AU
E2 154 ROSEN S C/AU
E3 779 --> ROSEN S D/AU
E4 8 ROSEN S D */AU

E5	1	ROSEN S D C/AU
E6	60	ROSEN S E/AU
E7	13	ROSEN S F/AU
E8	145	ROSEN S G/AU
E9	50	ROSEN S H/AU
E10	47	ROSEN S I/AU
E11	16	ROSEN S J/AU
E12	84	ROSEN S L/AU

=> s e3

L13 779 "ROSEN S D"/AU

=> e lee j k/au

E1	189	LEE J JACK/AU
E2	1	LEE J JR/AU
E3	4403 -->	LEE J K/AU
E4	4	LEE J K F/AU
E5	4	LEE J K H/AU
E6	1	LEE J K I/AU
E7	5	LEE J K J/AU
E8	1	LEE J K JERRY/AU
E9	3	LEE J K L/AU
E10	3	LEE J K M/AU
E11	3	LEE J K N/AU
E12	201	LEE J K P/AU

=> s e3

L14 4403 "LEE J K"/AU

=> e hemmerich s/au

E1	24	HEMMERICH RAINER/AU
E2	1	HEMMERICH ROLF H/AU
E3	129 -->	HEMMERICH S/AU
E4	90	HEMMERICH STEFAN/AU
E5	2	HEMMERICH W/AU
E6	1	HEMMERICK GEO/AU
E7	1	HEMMERICK PETER/AU
E8	20	HEMMERLE A/AU
E9	9	HEMMERLE A V/AU
E10	13	HEMMERLE ANKE/AU
E11	12	HEMMERLE C/AU
E12	8	HEMMERLE CHRISTINE/AU

=> s e3

L15 129 "HEMMERICH S"/AU

=> d his

(FILE 'HOME' ENTERED AT 08:53:43 ON 29 JUL 2005)

FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS, LIFESCI' ENTERED AT 08:54:06 ON 29 JUL 2005

L1	2838 S	GLYCOSYL (A)TRANSFERASE?
L2	594 S	(HUMAN OR MURINE) AND L1
L3	3 S	"GST4ALPHA"
L4	1 DUP REM	L3 (2 DUPLICATES REMOVED)
L5	7189603 S	CLON? OR EXPRESS? OR RECOMBINANT
L6	213 S	L2 AND L5
L7	1025751 S	COLON OR INTESTINE OR (CANCER(A)TISSUE?)
L8	21 S	L6 AND L7
L9	16 DUP REM	L8 (5 DUPLICATES REMOVED)
L10	258785 S	TRANSMEMBRANE
L11	8 S	L6 AND L10

L12 7 DUP REM L11 (1 DUPLICATE REMOVED)
E ROSEN S D/AU
L13 779 S E3
E LEE J K/AU
L14 4403 S E3
E HEMMERICH S/AU
L15 129 S E3

=> s l13 or l14 or l15

L16 5219 L13 OR L14 OR L15

=> s l2 and l16

L17 0 L2 AND L16

=> s l1 and l16

L18 0 L1 AND L16

=> s glycosyl (a) sulfotransferase?

L19 11 GLYCOSYL (A) SULFOTRANSFERASE?

=> dup rem l19

PROCESSING COMPLETED FOR L19

L20 7 DUP REM L19 (4 DUPLICATES REMOVED)

=> d 1-7 ibib ab

L20 ANSWER 1 OF 7 BIOTECHDS COPYRIGHT 2005 THE THOMSON CORP. on STN

ACCESSION NUMBER: 2003-11055 BIOTECHDS

TITLE: New mycobacterial peptide, useful for the manufacture of a
medicament for treating or preventing, or a diagnostic
reagent for identifying, mycobacterial infection;
vector plasmid-mediated recombinant protein gene transfer
and expression in host cell for use in recombinant vaccine
preparation against bacterium infection

AUTHOR: JAMES B W; MARSH P; HAMPSHIRE T

PATENT ASSIGNEE: MICROBIOLOGICAL RES AUTHORITY

PATENT INFO: WO 2003004520 16 Jan 2003

APPLICATION INFO: WO 2002-GB3052 4 Jul 2002

PRIORITY INFO: GB 2001-23993 5 Oct 2001; GB 2001-16385 4 Jul 2001

DOCUMENT TYPE: Patent

LANGUAGE: English

OTHER SOURCE: WPI: 2003-210338 [20]

AB DERWENT ABSTRACT:

NOVELTY - Isolated mycobacterial peptide (I) or its fragment, derivative
or variant, encoded by a mycobacterial gene, is new.

DETAILED DESCRIPTION - (I), encoded by a mycobacterial gene (II),
whose expression is induced or up-regulated under culture conditions that
are nutrient-starving and that maintain mycobacterial latency. The
conditions are obtainable by batch fermentation of a mycobacterium for at
least 20 days post-inoculation, when compared with culture conditions
that are not nutrient-starving and that support exponential growth of the
mycobacterium. INDEPENDENT CLAIMS are also included for the following:
(1) identifying the mycobacterial gene; (2) an inhibitor of (I); (3) an
antibody that binds to (I); (4) an attenuated mycobacterium in which a
gene has been modified, which renders the mycobacterium substantially
non-pathogenic; (5) an attenuated mycobacterial carrier comprising (I);
(6) a DNA plasmid; (7) an RNA sequence encoded by (II); (8) an RNA
vector; and (9) treating or preventing mycobacterial infection.

BIOTECHNOLOGY - Preferred Vector: The vector preferably comprises:
(a) the RNA sequence encoded by (II); and (b) an integration site for a
chromosome of a host cell. Preferred Inhibitor: The inhibitor is capable
of preventing or inhibiting the mycobacterial peptide from exerting its
native biological effect. It consists of: (a) an antibiotic capable of

targeting the induced or up-regulated mycobacterial gene or its gene product; or (b) an antisense or triplex-forming nucleic acid sequence that is complementary to at least part of the inducible or up-regulatable gene. The inhibitor is capable of inhibiting a protein comprising 2-nitropropane dioxygenase, acetyltransferase, oxidoreductase, transcriptional regulator, acyl transferase, UDP-glucose dehydrogenase, phosphoribosylglycinamide formyltransferase, glutathione reductase, dihydrolipoamide, transposase, proline iminopeptidase, prolyl aminopeptidase, quinolone efflux pump, glycine betaine transporter, phosphatidylethanolamine N-methyltransferase, chalcone synthase 2, **sulfotransferase**, **glycosyl transferase**, fumarate reductase flavoprotein, aminotransferase class-II pyridoxal-phosphate, bacteriophage HK97 prohead protease, penicillin-binding protein, fatty acyl-CoA racemase, nitrilotriacetate monooxygenase, histidine kinase response regulator or hydroxymethyl-dihydropterine pyrophosphokinase.

Preferred Gene: The gene to be modified has a wild-type coding sequence corresponding to a sequence comprising 210-4377 base pairs, fully disclosed in the specification. **Preferred Carrier:** The attenuated mycobacterial carrier is attenuated Salmonella, vaccine virus, fowlpox virus or Mycobacterium bovis (e.g. BCG strain). **Preferred Plasmid:** The DNA plasmid comprises: (a) a promoter; (b) a polyadenylation signal; and (c) a sequence that is the coding sequence of the mycobacterial gene. The promoter is cytomegalovirus and/or SV40 promoters. The polyadenylation signal consists of SV40 or bovine growth hormone polyadenylation signals. The DNA plasmid comprises 210-4377 bp. **Preferred Method:** Identifying the mycobacterial gene comprises: (a) culturing a first mycobacterium under culture conditions that are nutrient-starving and that maintain mycobacterial latency, where the conditions are obtainable by batch fermentation of a mycobacterium for at least 20 days post-inoculation; (b) culturing a second mycobacterium under culture conditions that are not-nutrient starving and that support exponential growth of the second mycobacterium; (c) obtaining first and second mRNA populations from the first and second mycobacteria, respectively, where the first mRNA population is obtained from the first mycobacterium and where the second mRNA is obtained from the second mycobacterium; (d) preparing first and second cDNA populations from the first and second mRNA populations, respectively, during which cDNA preparation, a detectable label is introduced into the cDNA molecules of the first and second cDNA populations; (e) isolating corresponding first and second cDNA molecules from first and second cDNA populations, respectively; (f) comparing relative amounts of label or corresponding signal emitted from the label present in the isolated first and second cDNA molecules; (g) identifying a greater amount of label or signal provided by the isolated first cDNA molecule than that provided by the isolated second cDNA molecule; and (h) identifying the first cDNA and the corresponding mycobacterial gene that is induced or up-regulated during culture of a mycobacterium under latency conditions. The corresponding first and second cDNA molecules are isolated from the first and second cDNA populations, respectively, by hybridization to an array plate containing immobilized amplified DNA sequences that have been generated from mycobacterial genomic DNA. The immobilized sequences are representative of each known gene of the mycobacterial genome. Each representative sequence is immobilized at an identified location on the plate. The first mycobacterium is cultured under culture conditions defined by a dissolved oxygen tension of less than 10%, preferably less than 7 or 5%, air saturation when measured at 37degreesC. It is harvested at least 30, preferably 40 days post-inoculation. The culture conditions are carbon-starving to the growth of the mycobacteria. A relative induction or up-regulation is identified by a relative 3-fold, preferably 4-fold increase in the amount of label or signal provided by the isolated first cDNA molecule over that provided by the isolated second cDNA molecule. Treating or preventing mycobacterial infection comprises administering to the patient the peptide, inhibitor, antibody, attenuated mycobacterium or microbial

carrier, DNA sequence or plasmid, or RNA sequence or vector.

ACTIVITY - Antibacterial. No biological data given.

MECHANISM OF ACTION - Vaccine.

USE - The peptide, inhibitor, antibody, attenuated mycobacterium or microbial carrier, DNA sequence or plasmid, or RNA sequence or vector is useful for the manufacture of a medicament for treating or preventing, or of a diagnostic reagent for identifying, mycobacterial infection (claimed).

ADMINISTRATION - The medicament is administered via intravenous, intraperitoneal or intranasal routes. No dosage given.

EXAMPLE - No relevant examples given. (440 pages)

L20 ANSWER 2 OF 7 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
DUPLICATE 1

ACCESSION NUMBER: 2002:281104 BIOSIS

DOCUMENT NUMBER: PREV200200281104

TITLE: Method of determining whether an agent modulates
glycosyl sulfotransferase-3.

AUTHOR(S): Bistrup, Annette [Inventor]; Rosen, Steven D. [Inventor,
Reprint author]; Tangemann, Kirsten [Inventor]; Hemmerich,
Stefan [Inventor]

CORPORATE SOURCE: San Francisco, CA, USA

ASSIGNEE: The Regents of the University of California

PATENT INFORMATION: US 6365365 20020402

SOURCE: Official Gazette of the United States Patent and Trademark
Office Patents, (Apr. 2, 2002) Vol. 1257, No. 1.
<http://www.uspto.gov/web/menu/patdata.html>. e-file.
CODEN: OGUPE7. ISSN: 0098-1133.

DOCUMENT TYPE: Patent

LANGUAGE: English

ENTRY DATE: Entered STN: 8 May 2002

Last Updated on STN: 8 May 2002

AB A novel human glycosylsulfotransferase expressed in high endothelial cells (GST-3) and polypeptides related thereto, as well as nucleic acid compositions encoding the same, are provided. The subject polypeptides and nucleic acid compositions find use in a variety of applications, including research, diagnostic, and therapeutic agent screening applications. Also provided are methods of inhibiting selectin mediated binding events and methods of treating disease conditions associated therewith, particularly by administering an inhibitor of at least one of GST-3 or KSGal6ST, or homologues thereof.

L20 ANSWER 3 OF 7 BIOTECHDS COPYRIGHT 2005 THE THOMSON CORP. on STN
DUPLICATE 2

ACCESSION NUMBER: 2001-06117 BIOTECHDS

TITLE: New **glycosyl-sulfotransferases**
(GST)-4-alpha, GST-4-beta and GST-6 for diagnostic and
therapeutic agent screening applications;
vector-mediated gene transfer, expression in host cell,
monoclonal antibody and transgenic animal for selectin
binding-inhibitor, drug screening and disease therapy,
diagnosis and gene therapy

AUTHOR: Rosen S D; Lee J K; Hemmerich S

PATENT ASSIGNEE: Univ. California

LOCATION: Oakland, CA, USA.

PATENT INFO: WO 2001006015 25 Jan 2001

APPLICATION INFO: WO 2000-US19741 19 Jul 2000

PRIORITY INFO: US 2000-593828 13 Jul 2000; US 1999-144694 20 Jul 1999

DOCUMENT TYPE: Patent

LANGUAGE: English

OTHER SOURCE: WPI: 2001-138471 [14]

AB A **glycosyl-sulfotransferase** (GST) (I) selected from
the group GST-4-alpha, GST-4-beta and GST-6, is claimed. Also claimed

are: a fragment of (I); a DNA (II) encoding (I); a DNA or its mimetic that hybridizes to (II) or its complementary sequence; an expression cassette (III) containing a transcriptional initiation region functional in an expression host and (II) under the transcriptional regulation of the transcriptional initiation region and a transcriptional termination region; a host cell (IV) containing (III); the cellular progeny of (IV); a method of producing (I); a monoclonal antibody that specifically binds to (I); and a non-human transgenic animal model for gene function, where the animal contains an introduced alteration in a gene encoding (I). (I) is useful for inhibiting a binding event between a selectin and a selectin ligand, which involves contacting the selectin with a non-sulfated selectin ligand. (II) encoding (I) is also useful in gene therapy to treat disorders such as acute or chronic inflammation and transplant tissue rejection and also for disease diagnosis. (44pp)

L20 ANSWER 4 OF 7 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 2001:427531 BIOSIS
DOCUMENT NUMBER: PREV200100427531
TITLE: Glycosyl sulfotransferase-3.
AUTHOR(S): Bistrup, Annette [Inventor, Reprint author]; Rosen, Steven D. [Inventor]; Hemmerich, Stefan [Inventor]
CORPORATE SOURCE: San Francisco, CA, USA
ASSIGNEE: The Regents of the University of California; Syntex, Inc., Palo Alto, CA, USA
PATENT INFORMATION: US 6265192 20010724
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (July 24, 2001) Vol. 1248, No. 4. e-file. CODEN: OGUPE7. ISSN: 0098-1133.
DOCUMENT TYPE: Patent
LANGUAGE: English
ENTRY DATE: Entered STN: 12 Sep 2001
Last Updated on STN: 22 Feb 2002

AB A novel human glycosylsulfotransferase expressed in high endothelial cells (GST-3) and polypeptides related thereto, as well as nucleic acid compositions encoding the same, are provided. The subject polypeptides and nucleic acid compositions find use in a variety of applications, including research, diagnostic, and therapeutic agent screening applications. Also provided are methods of inhibiting selectin mediated binding events and methods of treating disease conditions associated therewith.

L20 ANSWER 5 OF 7 BIOTECHDS COPYRIGHT 2005 THE THOMSON CORP. on STN
DUPLICATE 3

ACCESSION NUMBER: 2000-00104 BIOTECHDS
TITLE: Human and mouse **glycosyl-sulfotransferase**
-3 and related polynucleotides;
expression in mammalian host cell and antibody, used for
disease diagnosis and gene therapy
AUTHOR: Bistrup A; Rosen S D; Tangemann K; Hemmerich S
PATENT ASSIGNEE: Univ. California; Syntex
LOCATION: Oakland, CA, USA; Palo Alto, CA, USA.
PATENT INFO: WO 9949018 30 Sep 1999
APPLICATION INFO: WO 1999-US4316 26 Feb 1999
PRIORITY INFO: US 1998-190911 12 Nov 1998; US 1998-45284 20 Mar 1998
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: WPI: 1999-580442 [49]

AB **Glycosyl-sulfotransferase-3** (GST-3, 386 or 388 amino acids) present in other than its natural environment, is new. Also claimed are: a nucleic acid (2,032 or 1,893 bp) which encodes GST-3; an expression cassette under the control of initiation sequences and termination sequences; a host cell; a method of producing GST-3; a monoclonal antibody; a method for inhibiting the binding of a selectin

and a selectin ligand; a method of inhibiting a selectin mediated binding event in a mammalian host; a method of modulating a symptom of a disease condition associated with a selectin mediated binding event; a method of diagnosing a disease state related to the abnormal levels of a sulfotransferase chosen from GST-3 and KSGal6ST; a method of determining whether an agent is capable of modulating the activity of a sulfotransferase chosen from GST-3 and KSGal6ST; and a non-human transgenic animal model for *gst-3* gene function. The nucleic acid sequences, DNA probes and DNA primers derived from these, proteins and antibodies are useful in detecting homologs. The products are useful in the diagnosis of diseases associated with selectin binding interactions. (59pp)

L20 ANSWER 6 OF 7 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation on STN

ACCESSION NUMBER: 1998:793511 SCISEARCH

THE GENUINE ARTICLE: 130CC

TITLE: Cloning and functional characterization of a human **glycosyl sulfotransferase**, that is highly restricted to high endothelial venules and confers expression of the L-selectin recognition epitope 6-sulfo sialyl Lewis x.

AUTHOR: Hemmerich S (Reprint); Bistrup A; Bakhta S; Gunn M D; Kannagi R; Rosen S D

CORPORATE SOURCE: Roche Biosci, Palo Alto, CA USA; Univ Calif San Francisco, San Francisco, CA 94143 USA; Aichi Canc Res Inst, Nagoya, Aichi, Japan

COUNTRY OF AUTHOR: USA; Japan

SOURCE: GLYCOBIOLOGY, (NOV 1998) Vol. 8, No. 11, pp. 1112-1112. MA 29.

ISSN: 0959-6658.

PUBLISHER: OXFORD UNIV PRESS INC, JOURNALS DEPT, 2001 EVANS RD, CARY, NC 27513 USA.

DOCUMENT TYPE: Conference; Journal

LANGUAGE: English

REFERENCE COUNT: 0

ENTRY DATE: Entered STN: 1998

Last Updated on STN: 1998

L20 ANSWER 7 OF 7 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN DUPLICATE 4

ACCESSION NUMBER: 1999:17006 BIOSIS

DOCUMENT NUMBER: PREV199900017006

TITLE: Cloning and characterization of a human **glycosyl sulfotransferase** that is restricted to high endothelial venules and confers expression of the L-selectin recognition epitope 6-sulfo sialyl Lewis X.

AUTHOR(S): Bistrup, Annette [Reprint author]; Bakhta, Sunil; Tangemann, Kirsten; Lee, Jin Kyu; Gunn, Michael D.; Belov, Yevgeniy Y.; Kannagi, Reiji; Hemmerich, Stefan; Rosen, Steven D.

CORPORATE SOURCE: Univ. Calif., San Francisco, CA, USA

SOURCE: Molecular Biology of the Cell, (Nov., 1998) Vol. 9, No. SUPPL., pp. 124A. print.

Meeting Info.: 38th Annual Meeting of the American Society for Cell Biology. San Francisco, California, USA. December 12-16, 1998. American Society for Cell Biology.

CODEN: MBCEEV. ISSN: 1059-1524.

DOCUMENT TYPE: Conference; (Meeting)

Conference; Abstract; (Meeting Abstract)

LANGUAGE: English

ENTRY DATE: Entered STN: 20 Jan 1999

Last Updated on STN: 20 Jan 1999

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(FILE 'HOME' ENTERED AT 08:53:43 ON 29 JUL 2005)

FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS, LIFESCI' ENTERED AT 08:54:06 ON 29 JUL 2005

L1 2838 S GLYCOSYL (A)TRANSFERASE?
L2 594 S (HUMAN OR MURINE) AND L1
L3 3 S "GST4ALPHA"
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L8 21 S L6 AND L7
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L11 8 S L6 AND L10
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E ROSEN S D/AU
L13 779 S E3
E LEE J K/AU
L14 4403 S E3
E HEMMERICH S/AU
L15 129 S E3
L16 5219 S L13 OR L14 OR L15
L17 0 S L2 AND L16
L18 0 S L1 AND L16
L19 11 S GLYCOSYL (A) SULFOTRANSFERASE?
L20 7 DUP REM L19 (4 DUPLICATES REMOVED)

=> s l16 and l20

L21 3 L16 AND L20

=> d 1-3 ibib ab

L21 ANSWER 1 OF 3 BIOTECHDS COPYRIGHT 2005 THE THOMSON CORP. on STN

ACCESSION NUMBER: 2001-06117 BIOTECHDS

TITLE: New **glycosyl-sulfotransferases**
(GST)-4-alpha, GST-4-beta and GST-6 for diagnostic and
therapeutic agent screening applications;
vector-mediated gene transfer, expression in host cell,
monoclonal antibody and transgenic animal for selectin
binding-inhibitor, drug screening and disease therapy,
diagnosis and gene therapy

AUTHOR: Rosen S D; Lee J K; Hemmerich S
PATENT ASSIGNEE: Univ. California
LOCATION: Oakland, CA, USA.
PATENT INFO: WO 2001006015 25 Jan 2001
APPLICATION INFO: WO 2000-US19741 19 Jul 2000
PRIORITY INFO: US 2000-593828 13 Jul 2000; US 1999-144694 20 Jul 1999
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: WPI: 2001-138471 [14]

AB A **glycosyl-sulfotransferase** (GST) (I) selected from
the group GST-4-alpha, GST-4-beta and GST-6, is claimed. Also claimed
are: a fragment of (I); a DNA (II) encoding (I); a DNA or its mimetic
that hybridizes to (II) or its complementary sequence; an expression
cassette (III) containing a transcriptional initiation region functional
in an expression host and (II) under the transcriptional regulation of
the transcriptional initiation region and a transcriptional termination
region; a host cell (IV) containing (III); the cellular progeny of (IV);

a method of producing (I); a monoclonal antibody that specifically binds to (I); and a non-human transgenic animal model for gene function, where the animal contains an introduced alteration in a gene encoding (I). (I) is useful for inhibiting a binding event between a selectin and a selectin ligand, which involves contacting the selectin with a non-sulfated selectin ligand. (II) encoding (I) is also useful in gene therapy to treat disorders such as acute or chronic inflammation and transplant tissue rejection and also for disease diagnosis. (44pp)

L21 ANSWER 2 OF 3 BIOTECHDS COPYRIGHT 2005 THE THOMSON CORP. on STN

ACCESSION NUMBER: 2000-00104 BIOTECHDS

TITLE: Human and mouse **glycosyl-sulfotransferase**
-3 and related polynucleotides;
expression in mammalian host cell and antibody, used for
disease diagnosis and gene therapy

AUTHOR: Bistrup A; **Rosen S D**; Tangemann K; **Hemmerich S**

PATENT ASSIGNEE: Univ. California; Syntex

LOCATION: Oakland, CA, USA; Palo Alto, CA, USA.

PATENT INFO: WO 9949018 30 Sep 1999

APPLICATION INFO: WO 1999-US4316 26 Feb 1999

PRIORITY INFO: US 1998-190911 12 Nov 1998; US 1998-45284 20 Mar 1998

DOCUMENT TYPE: Patent

LANGUAGE: English

OTHER SOURCE: WPI: 1999-580442 [49]

AB **Glycosyl-sulfotransferase**-3 (GST-3, 386 or 388 amino acids) present in other than its natural environment, is new. Also claimed are: a nucleic acid (2,032 or 1,893 bp) which encodes GST-3; an expression cassette under the control of initiation sequences and termination sequences; a host cell; a method of producing GST-3; a monoclonal antibody; a method for inhibiting the binding of a selectin and a selectin ligand; a method of inhibiting a selectin mediated binding event in a mammalian host; a method of modulating a symptom of a disease condition associated with a selectin mediated binding event; a method of diagnosing a disease state related to the abnormal levels of a sulfotransferase chosen from GST-3 and KSGal6ST; a method of determining whether an agent is capable of modulating the activity of a sulfotransferase chosen from GST-3 and KSGal6ST; and a non-human transgenic animal model for *gst-3* gene function. The nucleic acid sequences, DNA probes and DNA primers derived from these, proteins and antibodies are useful in detecting homologs. The products are useful in the diagnosis of diseases associated with selectin binding interactions. (59pp)

L21 ANSWER 3 OF 3 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation on STN

ACCESSION NUMBER: 1998:793511 SCISEARCH

THE GENUINE ARTICLE: 130CC

TITLE: Cloning and functional characterization of a human **glycosyl sulfotransferase**, that is highly restricted to high endothelial venules and confers expression of the L-selectin recognition epitope 6-sulfo sialyl Lewis x.

AUTHOR: **Hemmerich S (Reprint)**; Bistrup A; Bakhta S; Gunn M D; Kannagi R; **Rosen S D**

CORPORATE SOURCE: Roche Biosci, Palo Alto, CA USA; Univ Calif San Francisco, San Francisco, CA 94143 USA; Aichi Canc Res Inst, Nagoya, Aichi, Japan

COUNTRY OF AUTHOR: USA; Japan

SOURCE: GLYCOBIOLOGY, (NOV 1998) Vol. 8, No. 11, pp. 1112-1112. MA 29.

ISSN: 0959-6658.

PUBLISHER: OXFORD UNIV PRESS INC, JOURNALS DEPT, 2001 EVANS RD, CARY,

NC 27513 USA.
DOCUMENT TYPE: Conference; Journal
LANGUAGE: English
REFERENCE COUNT: 0
ENTRY DATE: Entered STN: 1998
Last Updated on STN: 1998

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E LEE J K/AU
L14 4403 S E3
E HEMMERICH S/AU
L15 129 S E3
L16 5219 S L13 OR L14 OR L15
L17 0 S L2 AND L16
L18 0 S L1 AND L16
L19 11 S GLYCOSYL (A) SULFOTRANSFERASE?
L20 7 DUP REM L19 (4 DUPLICATES REMOVED)
L21 3 S L16 AND L20

	L #	Hits	Search Text
1	L1	14	glycosyl adj sulfotransferase\$3
2	L2	0	"GST4alpha"
3	L3	24	"gst4##"
4	L4	49880 1	human or murine
5	L5	8	l3 same l4
6	L6	75449 0	clon\$3 or express\$3 or recombinant
7	L7	128	("l14" or l3) same l6
8	L8	5	l3 same l6
9	L9	124	"l14" same l6
10	L10	5283	"l1" same l6
11	L11	7	l1 same l6
12	L12	10	l8 or l11
13	L13	29989 0	ROSEN LEE HEMMERICH
14	L14	14	l1 and l13
15	L15	12	l3 and l13

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1	20041209	101	US 20040249131 A1	Mycobacterial sulfation pathway proteins and methods of use thereof
2	20040923	140	US 20040185546 A1	Novel glycosyl sulfotransferases GST-4alpha, GST-4beta, & GST-6
3	20030925	101	US 20030180321 A1	Mycobacterial sulfation pathway proteins and methods of use thereof
4	20030605	98	US 20030104001 A1	Mycobacterial sulfation pathway proteins and methods of use thereof
5	20021107	36	US 20020164748 A1	Glycosyl sulfotransferase-3
6	20011213	27	US 20010051370 A1	Glycosyl sulfotransferase-3
7	20050308	97	US 6863895 B2	Mycobacterial sulfation pathway proteins and methods of use thereof
8	20050222	112	US 6858213 B2	Mycobacterial sulfation pathway proteins and methods of use thereof
9	20050208	135	US 6852518 B1	Glycosyl sulfotransferases GST-4.alpha., GST-4.beta., and GST-6
10	20050118	38	US 6844175 B2	Methods of inhibition using glycosyl sulfotransferase-3
11	20020528	24	US 6395882 B1	Selectin ligands
12	20020430	18	US 6380371 B1	Endoglycan: a novel protein having selectin ligand and chemokine presentation activity
13	20020402	39	US 6365365 B1	Method of determining whether an agent modulates glycosyl sulfotransferase-3
14	20010724	27	US 6265192 B1	Glycosyl sulfotransferase-3

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1	20050630	33	US 20050144690 A1	Inbred corn line PHEHG
2	20050630	32	US 20050144689 A1	Inbred com line PHACV
3	20050630	34	US 20050144688 A1	Inbred corn line PHAR1
4	20050630	34	US 20050144687 A1	Inbred corn line PHCPR
5	20050602	35	US 20050120439 A1	Inbred corn line PHADA
6	20050526	35	US 20050114953 A1	Inbred corn line PHCMV
7	20050526	35	US 20050114952 A1	Inbred corn line PHCND
8	20050526	35	US 20050114951 A1	Inbred corn line PHC77
9	20050526	30	US 20050114945 A1	Inbred corn line PHCK5
10	20050217	22	US 20050037418 A1	Method and apparatus for assaying a drug candidate to estimate a pharmacokinetic parameter associated therewith
11	20050210	172	US 20050031643 A1	Microorganisms for therapy
12	20041007	23	US 20040197785 A1	Method for quantitative measurement of gene expression for indentifying individuals at risk for bronchogenic carcinoma
13	20040923	140	US 20040185546 A1	Novel glycosyl sulfotransferases GST-4alpha, GST-4beta, & GST-6

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14	20040722	293	US 20040142335 A1	Method for determining skin stress or skin ageing in vitro
15	20040212	56	US 20040029149 A1	Human metabolic models and methods
16	20030911	34	US 20030170263 A1	Expression system
17	20030501	78	US 20030082511 A1	Identification of modulatory molecules using inducible promoters
18	20030213	33	US 20030031681 A1	Combined growth factor-deleted and thymidine kinase-deleted vaccinia virus vector
19	20020214	22	US 20020019019 A1	Method and apparatus for assaying a drug candidate to estimate a pharmacokinetic parameter associated therewith
20	20050208	135	US 6852518 B1	Glycosyl sulfotransferases GST-4.alpha., GST-4.beta., and GST-6
21	20041026	21	US 6808938 B2	Method and apparatus for assaying a drug candidate to estimate a pharmacokinetic parameter associated therewith
22	20000711	14	US 6088277 A	Read only memory capable of realizing a high-speed read operation
23	19861216	22	US 4630188 A	Multi-zone ramp system for digital pulse generator and large scale integrated chip embodying the same
24	19821102	9	US 4357584 A	Acoustic wave devices

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